

Physiology Summary

First Year

By

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Lecturer Physiology

Ain Shams University

2017

VISIT...

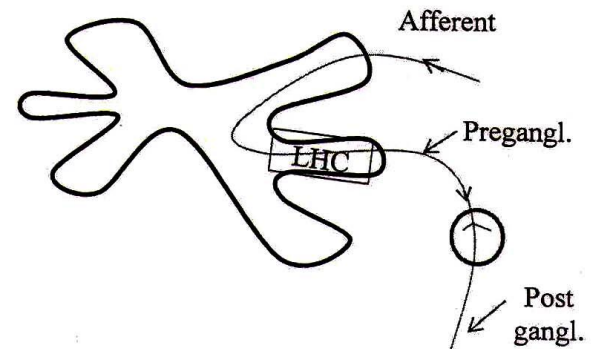
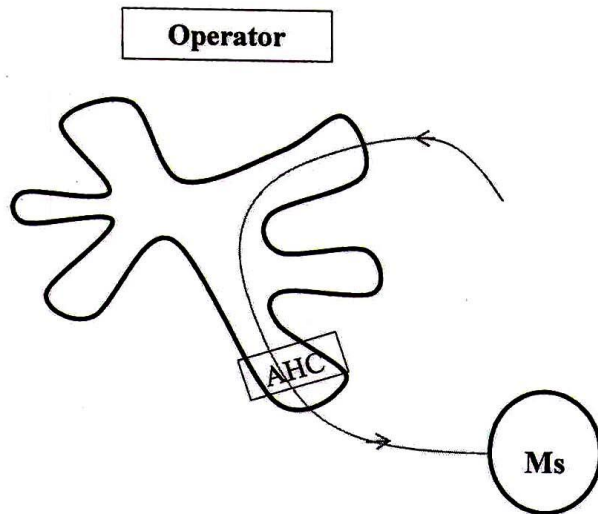
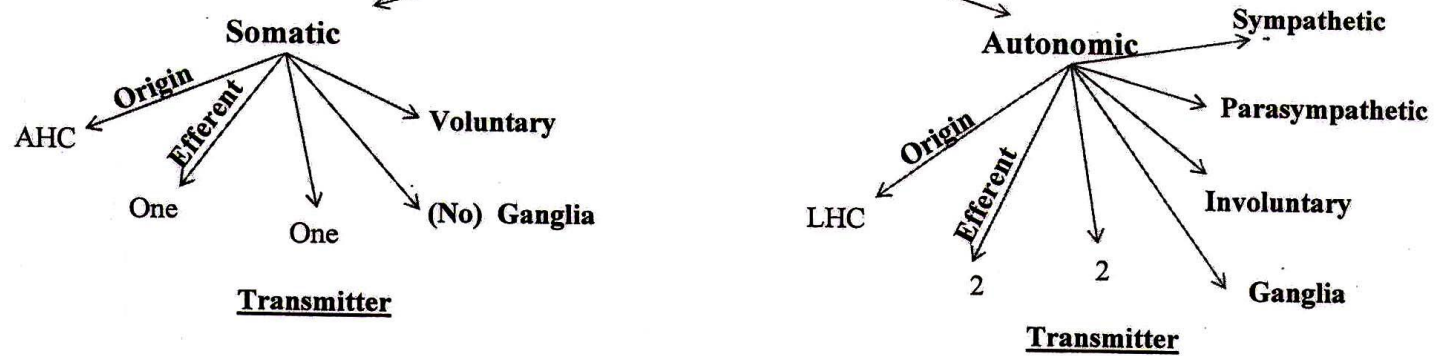
LANZAROTE
Caliente.COM

ملخص لكل أجزاء النظري في ١٨٧ صفحة فقط ويشمل :

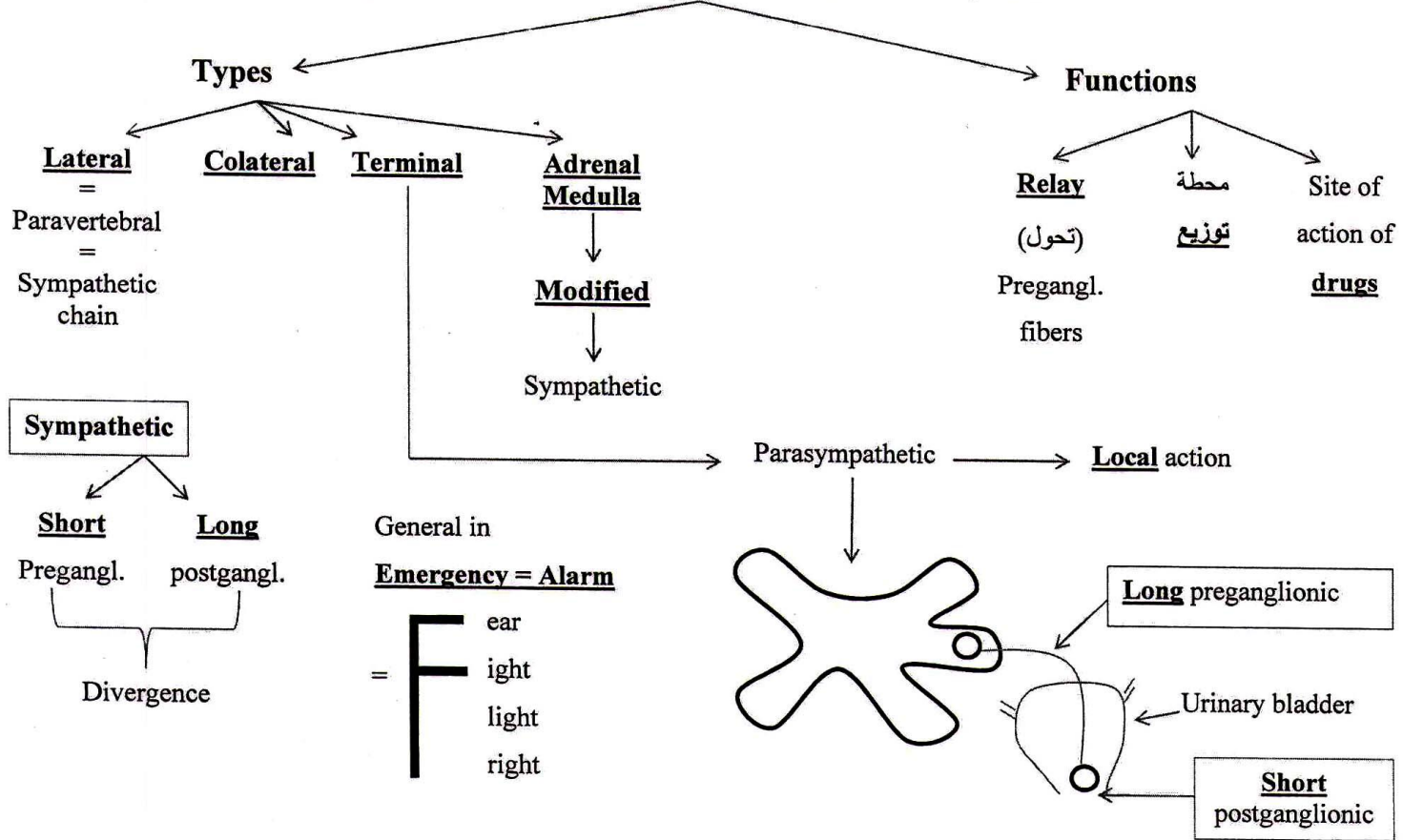
- 1) **Autonomic:** 9 pages
- 2) **Nerve:** 4 pages
- 3) **Muscle :** 15 page
- 4) **Blood:** 16 pages
- 5) **CVS:** 83 pages
- 6) **Respiration:** 35 pages
- 7) **Biophysics:** 9 page
- 8) **Biostatistics:** 8 Pages
- 9) **Collection:** 8 pages

Autonomic

د. محمد فايز Nervous System



Ganglia (Nerve Cells Outside CNS) د. محمد فايز



	Sympathetic	Parasympathetic
Origin	LHC of Thoracolumbar segments 	Cranio sacral
<u>Actions</u> General	Sympathetic tone ↓ Mild vasoconstriction ↓ Average BP (120/80)	Parasymp. Tone ↓ HR from 110 to 70 bpm
Head, Neck	1) Eye 2) Saliva → viscid → contrac. Myoepithelial cells around acini 3) BV → VC 4) Sweating Horner Syndrome	العكس (Symp.) Parasymp. يساعد في (cooperation) Saliva

	Sympathetic	Parasympathetic
Respiratory	Broncho dilatation VC pulmonary artery	العكس
CVS	A) ↑ Cardiac Excitability Contraction force Rhythmicity Conduction ملاحظة هامة ↑ HR ↑ Force of contraction Chronotropic Inotropic B) Vascular: VD coronary Direct Indirect	العكس → Vagus العكس

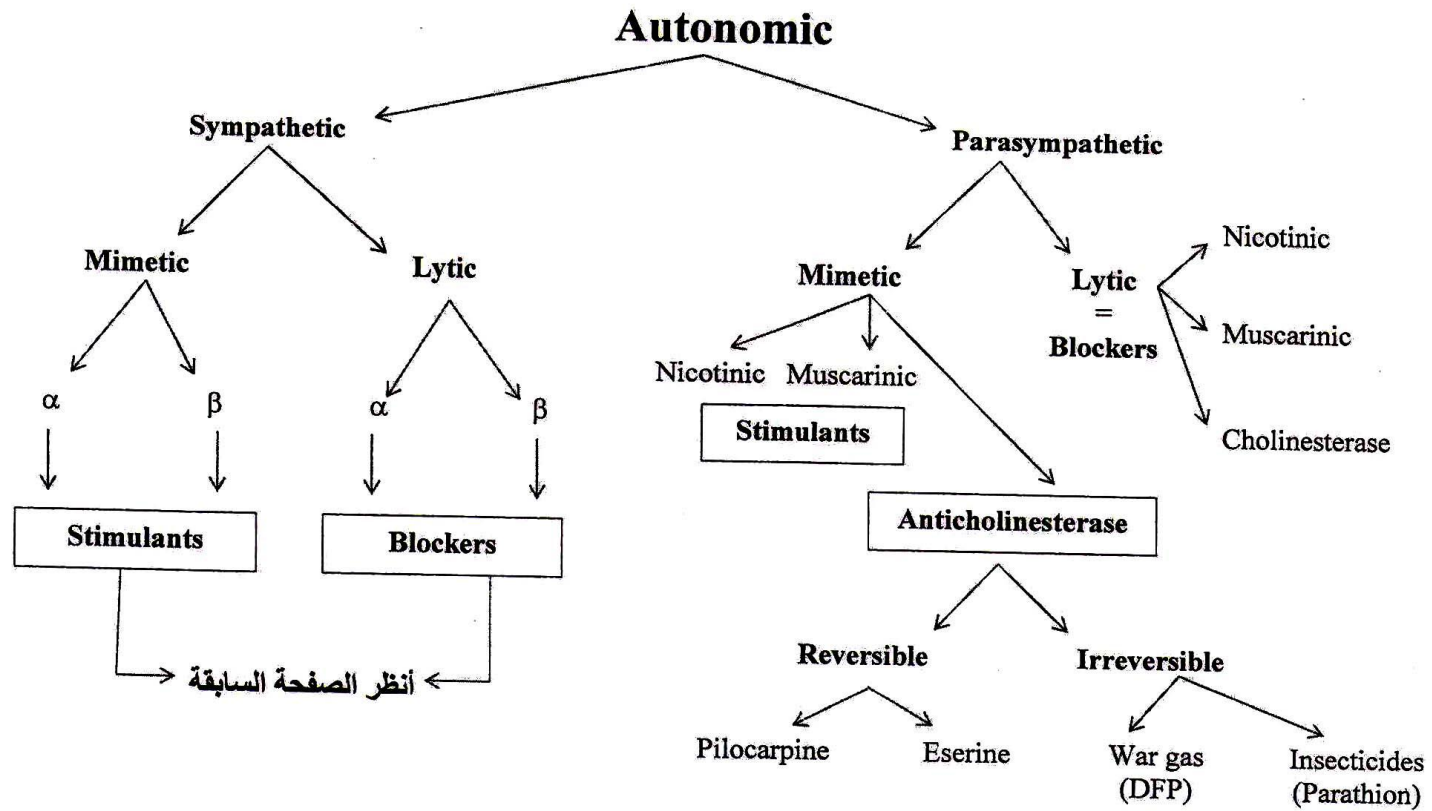
	Sympathetic	Parasympathetic
<u>GIT</u>	Greater splanchnic	Vagus
1. Stomach	Relax wall, contract sphincter	Digestion, Motility العكس هيعمل
2. Liver	Glycogenolysis	--
3. Gall bladder	Relax wall, contract sphincter	Bile العكس هيفرز
4. Spleen	Contraction	---
5. Kidney	↑ Renin secretion	
6. BV	VC	VD
<u>Pelvis</u>	← Lesser splanchnic →	← Pelvic nerve →
1. Rectum	Relax wall, contract internal sphincter	Defecation العكس هيعمل
2. Urinary bladder	Relax wall, contract internal sphincter	Micturition العكس هيعمل
3. Genitalia	Ejaculation (النهاية)	Erection (البداية)
Organs only supplied by	S kin keletal ms VC pleen upra renal + Ventricles + Pupilodilator	Pupilo constrictor Oesophagus Gastric Glands

Chemical Transmitters

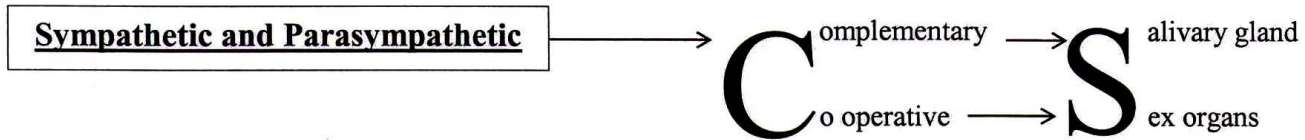
	Acetyl choline	Noradrenaline
Synthesis	Choline + Acetyl CoA ↓ Acetyl choline	Tyrosine ↓ DOPA ↓ Dopamine ↓ Noradrenaline → Adrenaline
Site of release	• <u>All preganglionic.</u> • <u>All post ganglionic parasymp.</u> • <u>Some post ganglionic symp.</u> (sweat gl., BV of coronary, skeletal).	• Post ganglionic sympathetic. • Adrenal medulla (20%).
Actions	Parasympathetic (أنظر ما سبق)	Sympathetic (أنظر ما سبق)
Receptor	أنظر الصفحة القادمة	
Removal	← Active • Cholinesterase - True - Specific - RBCs - Pseudo - Not Specific - Plasma	Reuptake → • MAO. • COMT. • VMA.

Autonomic Receptors

	Acetyl choline		Nor adrenaline			
Site	Nicotinic	Muscarinic	$\alpha 1$	$\alpha 2$	$\beta 1$	$\beta 2$
	Nn ↙ ↘ Nm					
	* NMJ * Ganglia * Adrenal medulla	* M1: Brain * M2: Heart * M3: Smooth ms	* Wall of BV * Spleen * Sphincters	* Presynaptic * Platelets	* Heart * JGA * Fat tissue	Smooth ms of BV Bronchi
Structure	Ionotropic	← Serpentine →				
Mechanism	Na ⁺ influx → Depolarisation		↑ Ca ⁺⁺	↓ c.AMP	↑ c.AMP	↑ c.AMP
Stimulated by	* Acetyl choline * Nicotine small dose	* Acetyl choline * Muscarine	← Adrenaline NA يمسك في $\alpha 1$ اقوى * Phenyl ephrine	فيهم NA , ولكن	يمسك * Propranolol	→ كلهم يمسك Adrenaline في $\beta 2$ اقوى * Salbutamol
Blocked by	* Nicotine large dose * Curare * Hexamethonium	* Atropine إستخداماته: 1. Preanesthesia 2. Fundus 3. colic علاج	Phentolamine	Yohimbine	Inderal	Butoxamine

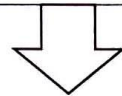


د. محمد فايز Notes



Exercise

- ↑ Lipolysis.
- VD of skeletal ms arterioles.

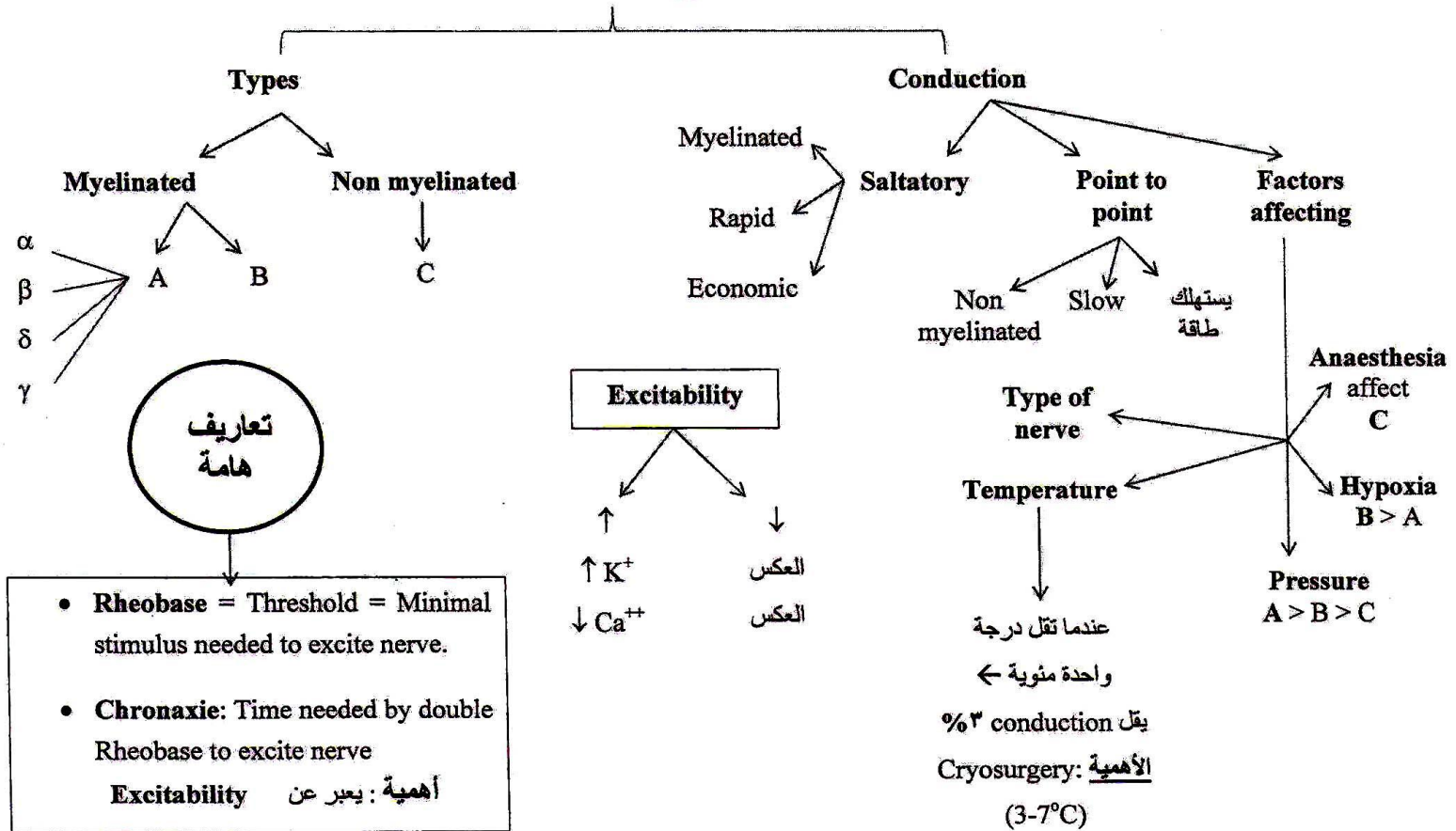


Orbelli effect

9a

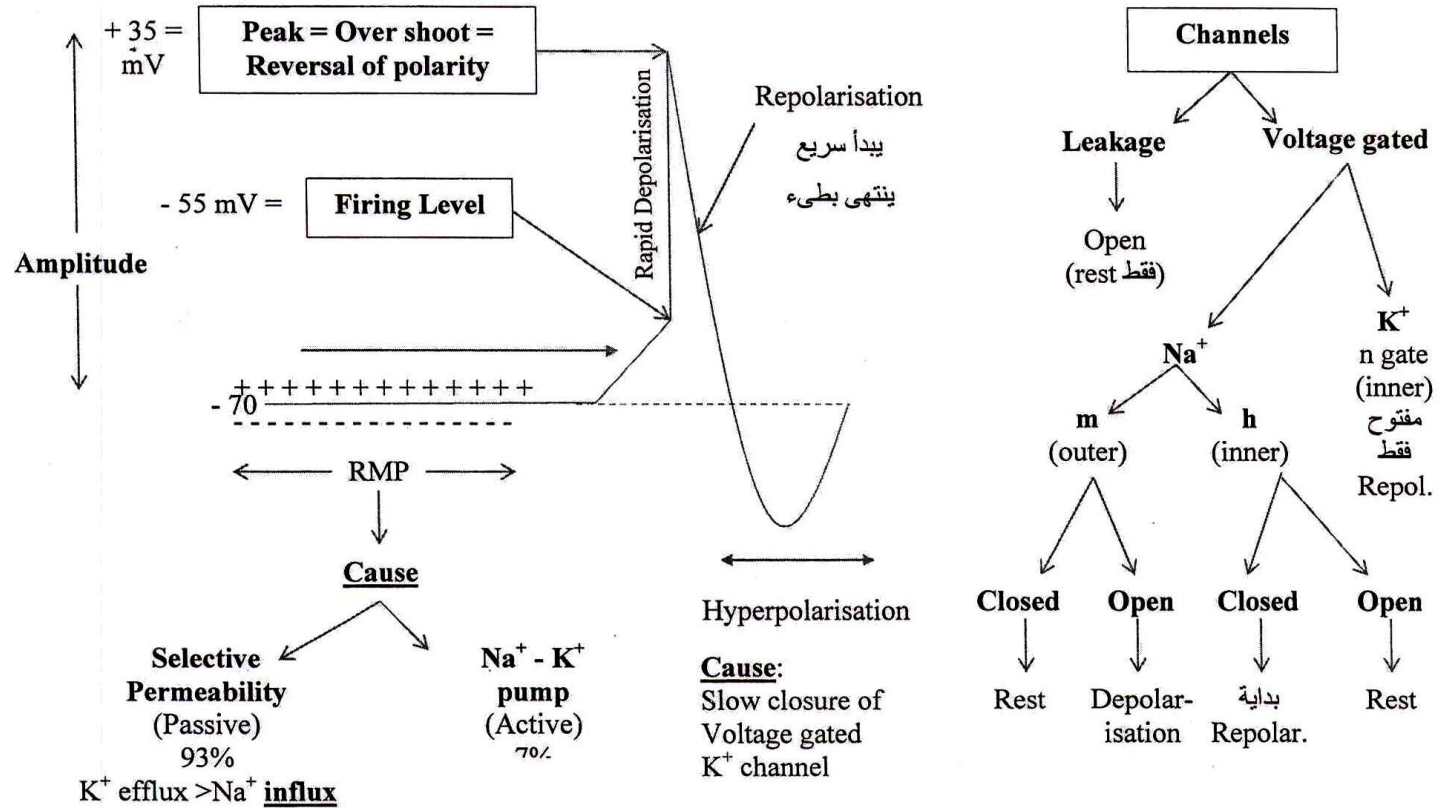
Nerve

د. محمد فايز Nerve

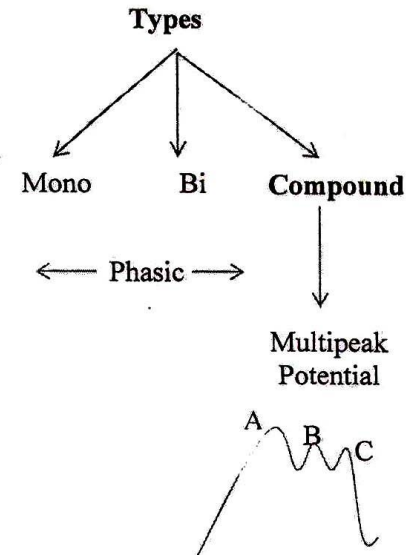
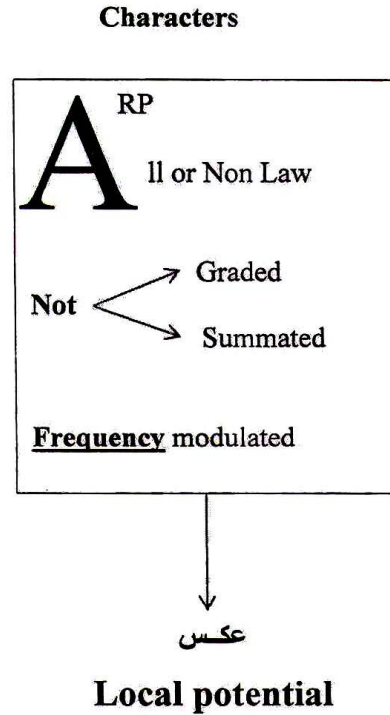
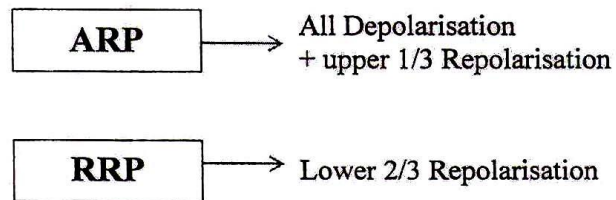
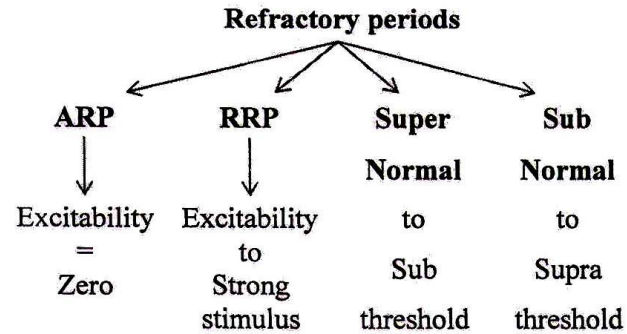


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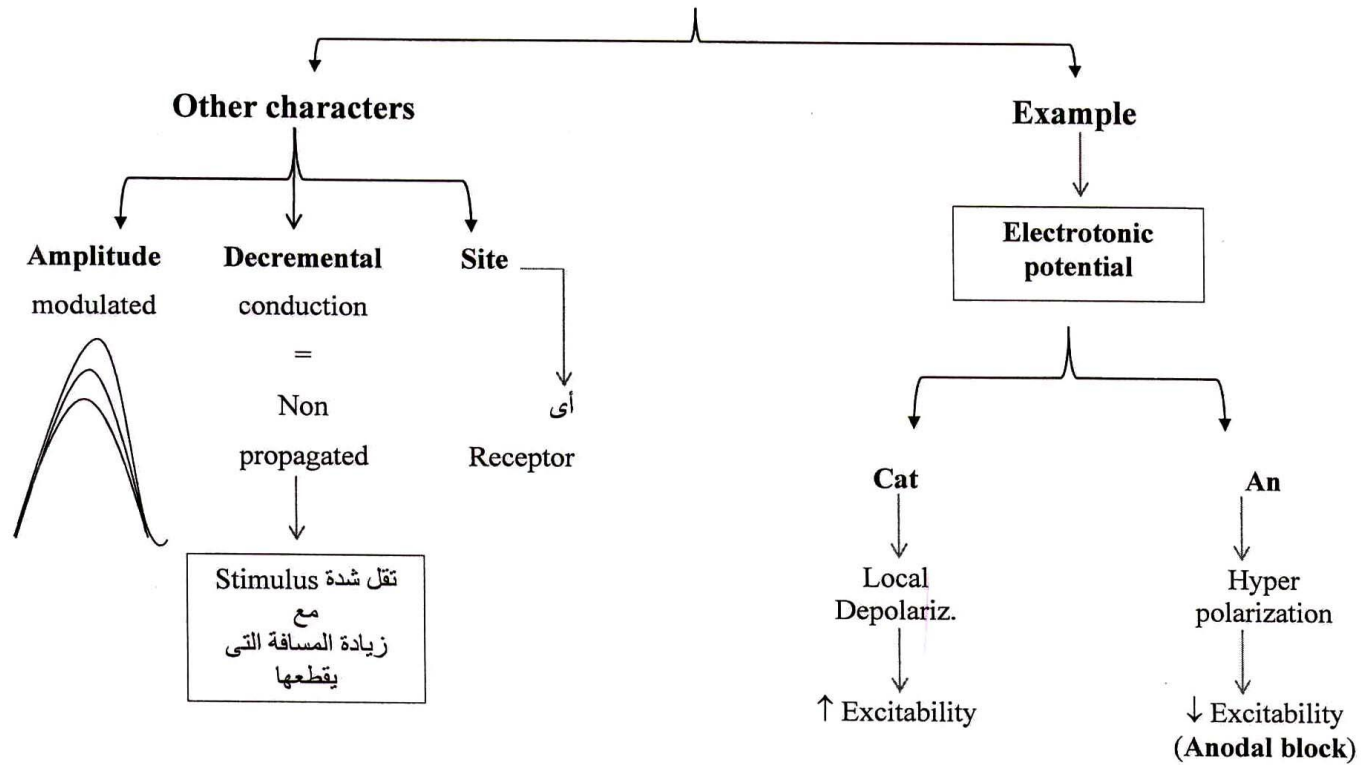
د. محمد فايز Action Potential



د. محمد فايز Action Potential



د. محمد فايز Local Potential



د. محمد فايز Effects obtained from different stimuli applied on mixed nerves

	Subthreshold	Threshold	Supathreshold	Maximal	Supramaximal
Excitability	--	Small number of nerve fibers	Great number of nerve fibers	All nerve fibers	Each nerve fiber obeys all or none law
Potential changes recorded	--	Small	Large	Maximal	No further ↑

Potential nerves recorded represent algebraic summation of "all or none" action potential of many nerve fibers

12 a

Muscle

د. محمد فايز (Excitation contraction coupling) Muscle

Action potential opens voltage gated Ca^{++} channels (Nerve)

↓
 Ca^{++} influx

↓
Rupture of Ach. Vesicles

↓
Release of Ach.

↓
Ach. Binds to nicotinic receptors (**MEP**)

↓
AP. Reaches sarcolemma (MS membrane)

↓
Pass through T. Tubes

↓
Opens **DHP Receptor** (voltage sensor)

↓
Opens **ryanodine** Ca^{++} channels (ligand gated) on SR

↓
 Ca^{++} **release** from cisterna (SR)

↓
 Ca^{++} binds to **troponin C**

↓
Removes tropomyosin which covers active sites on actin

↓
Cross bridge cycling

Neuro MS Transmission

1. Unidirectional
2. Delay.
3. Fatigue.
4. Affected by :

Stimulatory

A. Nicotine
small dose

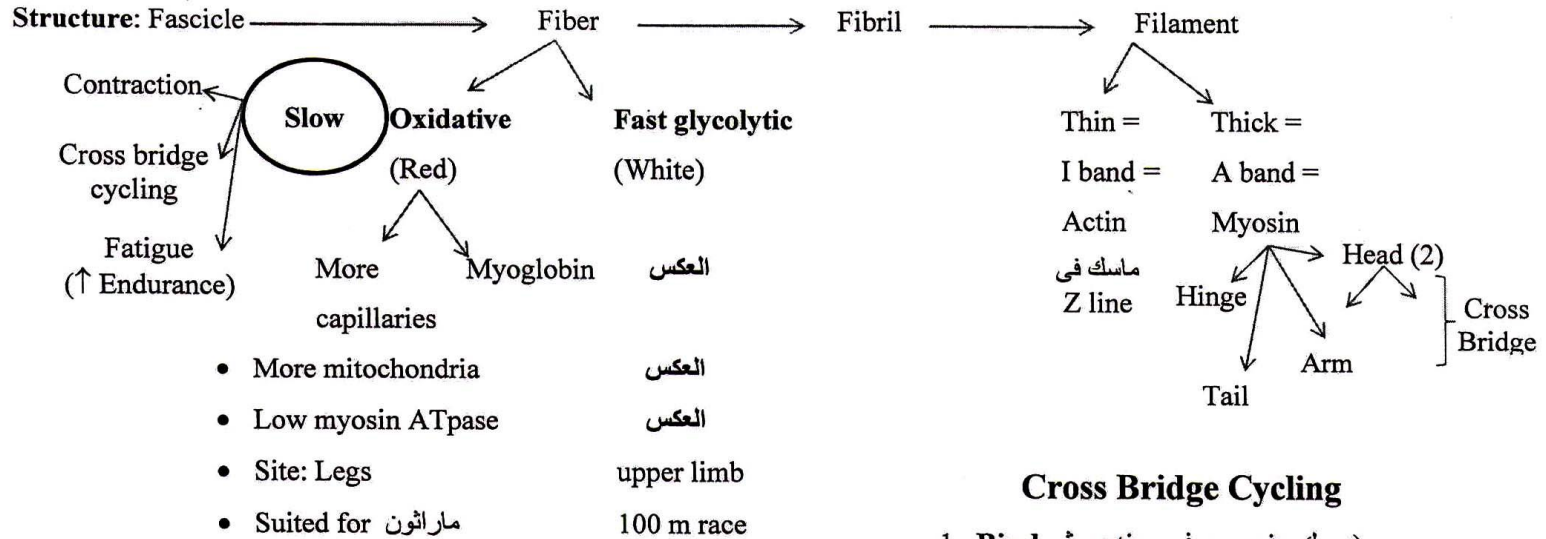
B. Anticholin-
esterase

Inhibitory

العكس
(+)

-Succinyl choline.
-Botulinum toxins

د. محمد فايز Muscle



Notes:

1. Walk along theory:

Ca^{++} binds to troponin C → remove tropomyosin

→ Cross bridge cycling.

2. Contracture: continuous contraction without relaxation.

Cross Bridge Cycling

1. Bind (يمسك myosin في actin ثم)

(يشده جوه ناحية Sarcomere center)

2. Bend (power stroke).

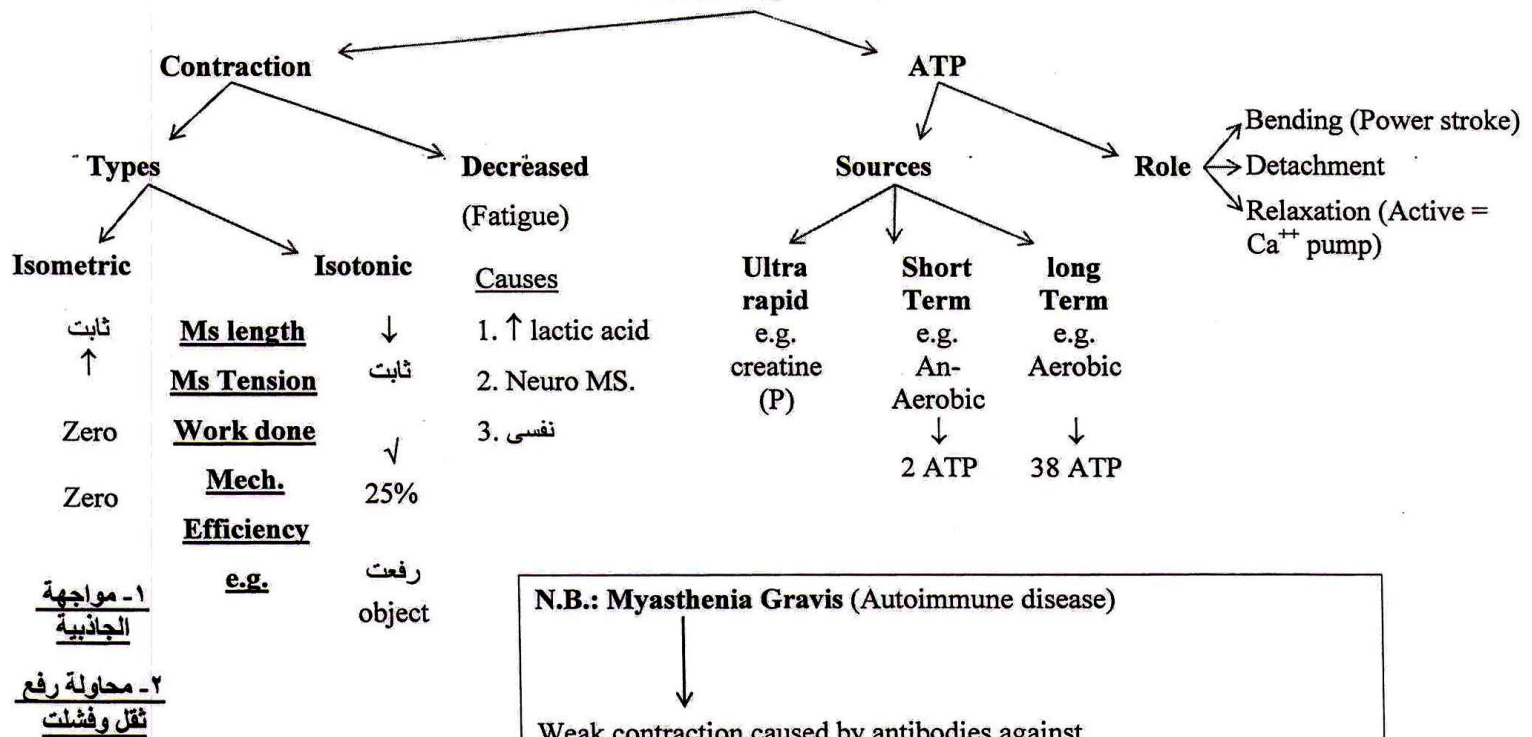
3. Detachment:

إذا لم ينفصل myosin head عن Actin

← Rigor mortis (بعد الوفاة)

4. Recycling.

د. محمد فايز Muscle



N.B.: Myasthenia Gravis (Autoimmune disease)

Weak contraction caused by antibodies against

Some Nicotinic receptors at MEP.

Treated by: Neostigmine

د. محمد فايز Muscle

Electrical events

RMP: -90 mv

Firing level: -40 mv

Peak: +40 mv

Amplitude: 130 mv

Action potential precedes (يسبق)

Contraction by 2 msec.

Types of Heat

1. Resting
2. Shortening
3. Maintenance
4. Recovery.

O₂ debt

Def. Extra O₂ paid during recovery (Exercise)

Types (MCQ هام)

alactic

2-3 minutes

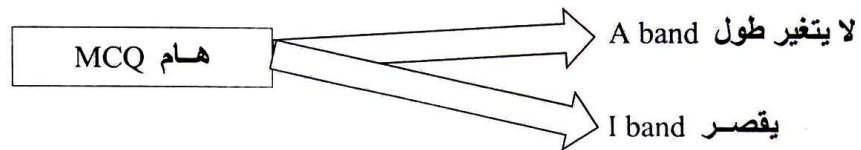
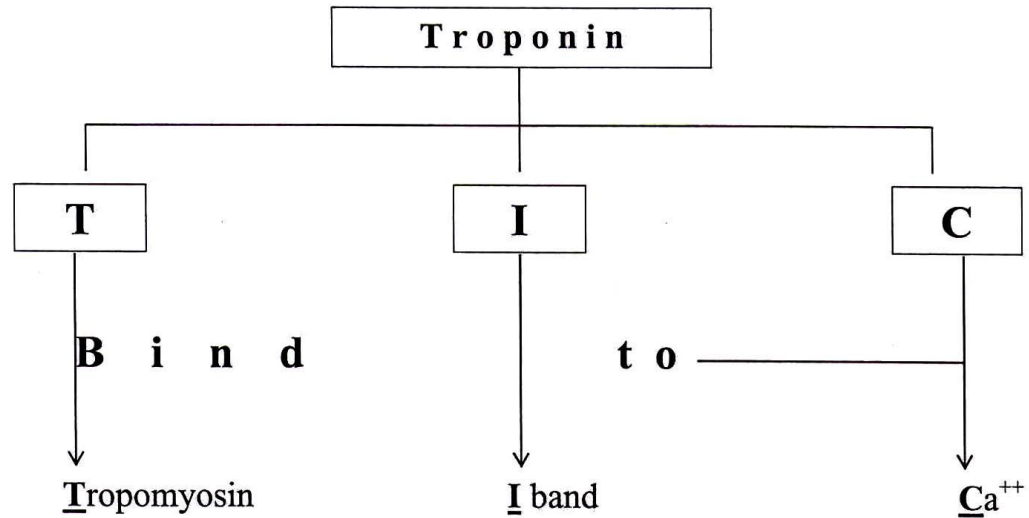
1. Replenish ATP
2. Restore CP
3. Rebind to myoglobin

Lactic

1 hour

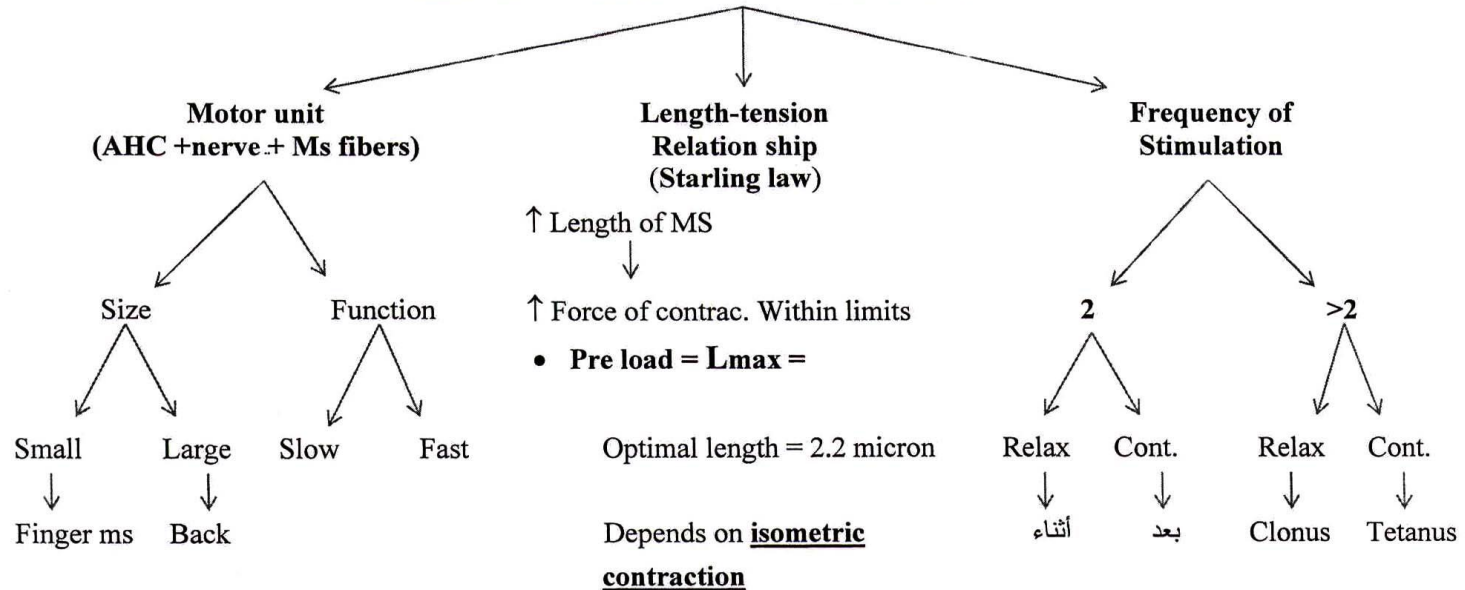
1. Krebs
2. glucose in liver يتحول إلى
3. Stored as glycogen in MS

د. محمد فايز Notes on Excitation Contraction Coupling



169

د. محمد فايز Gradation/. of skeletal ms contraction



- After load:
- V_{max} at zero after load
- Depends on isotonic contraction.

N.B.: Stair case phenomena

↑ Ca⁺⁺ influx →
 ↑ Force of contraction gradual
 كأنك طالع سلم درجة درجة

Simple Muscle Twitch (SMT) د. محمد فايز



Single stimulus

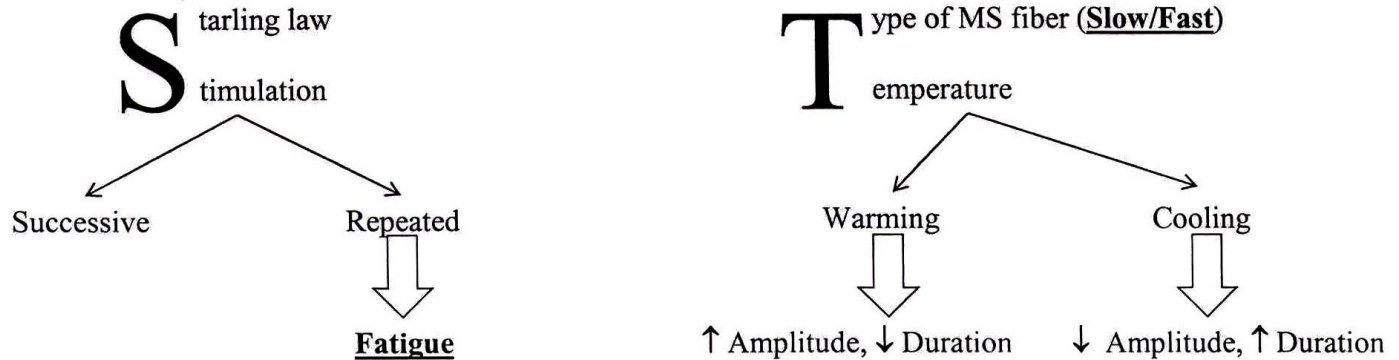


Single contraction (0.04 sec)



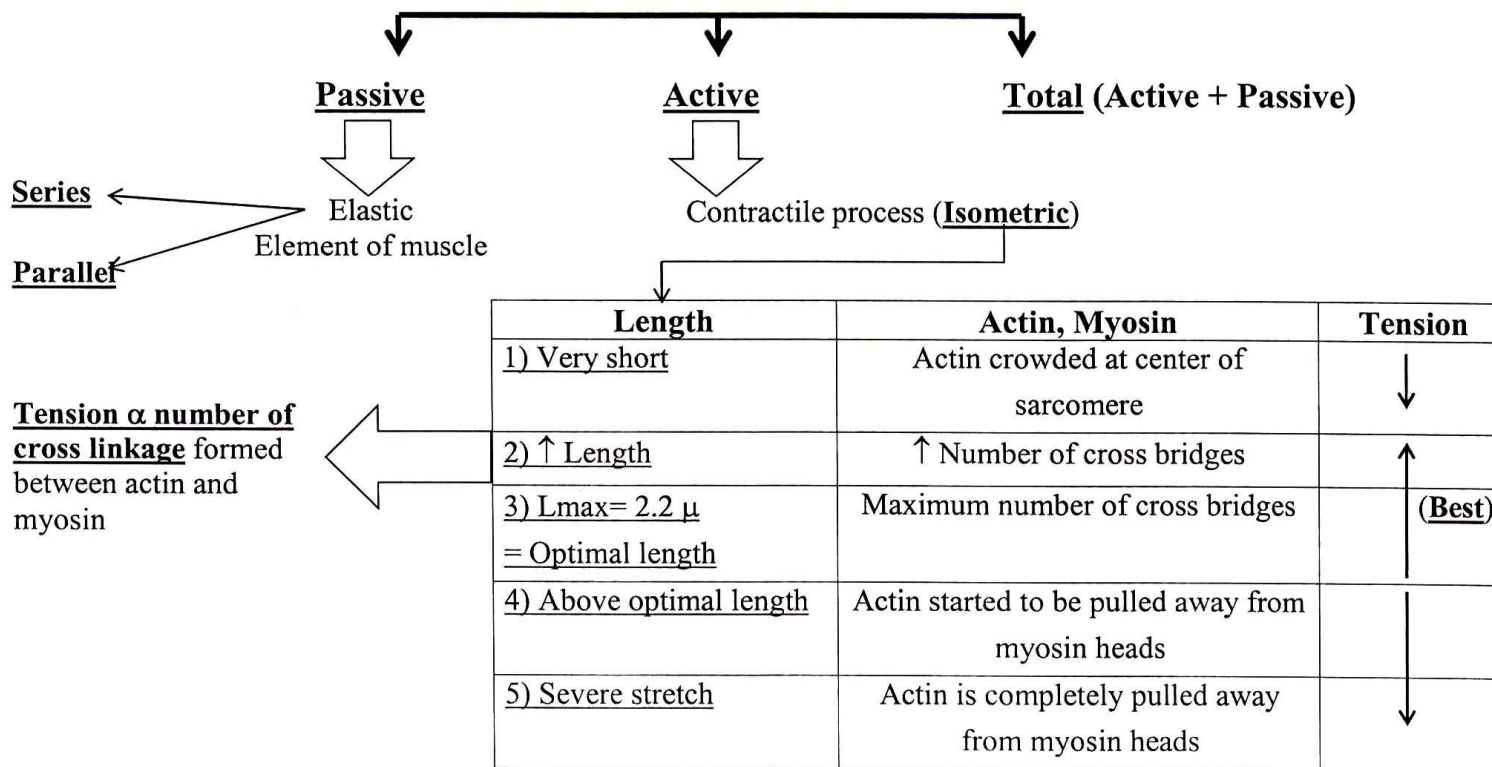
Single Relaxation (0.05 sec)

Factors affecting SMT



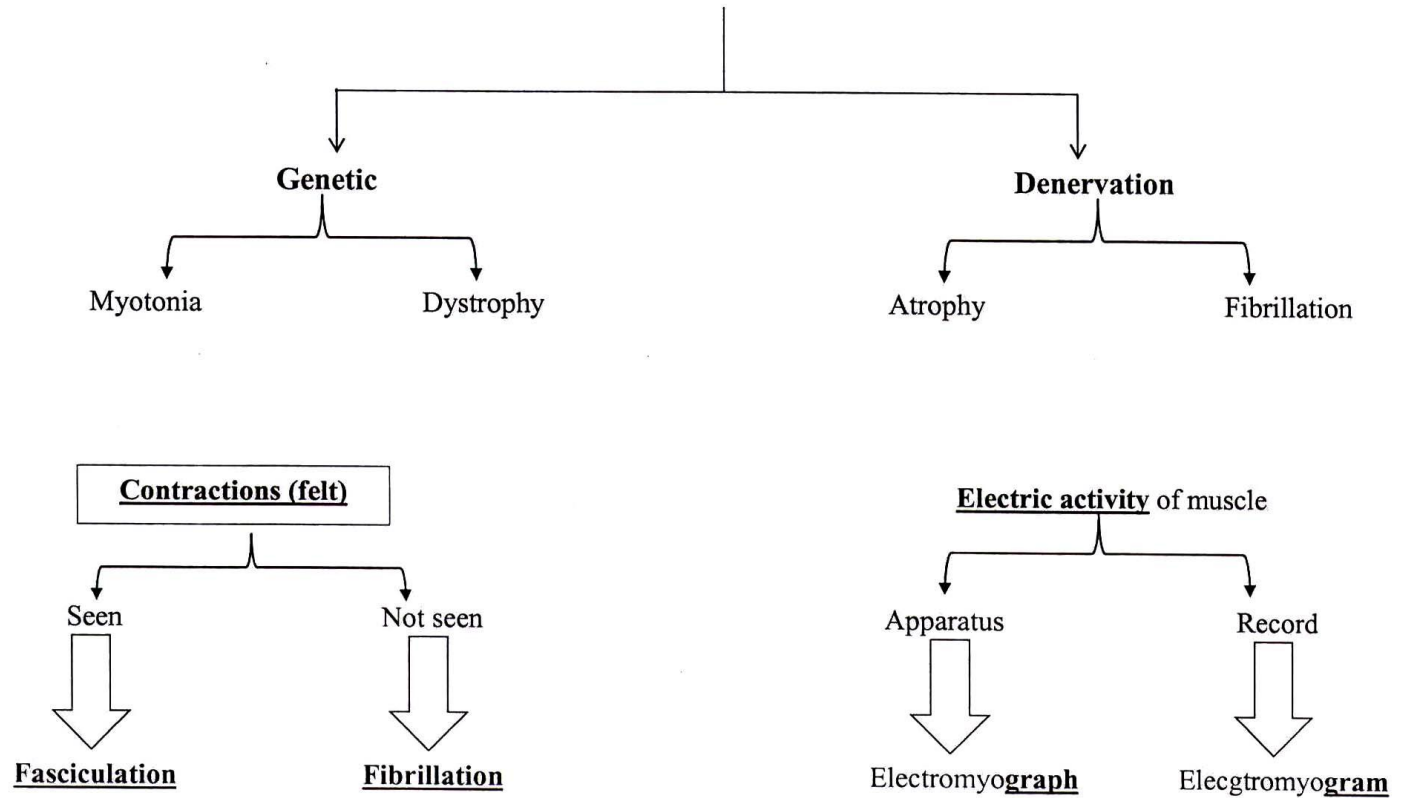
17a

د. محمد فايز Length-Tension Relationship



176

Muscle Diseases د. محمد فايز

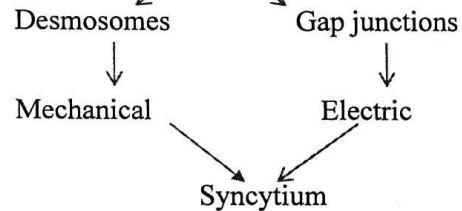


17c

د. محمد فايز Cardiac muscle

Structure

1. Has intercalated discs



2. Poor developed Sr.

3. Skeletal ms عندها

Actin
Myosin
Z line
Troponin
Tropomyosin

Excitation Contraction Coupling

نفس خطوات

Skeletal ms

مع فارق هام

يعتمد القلب على

ECF Ca^{++}

(Trigger = induced Ca^{++})

Which stimulates Ca^{++} release

From SR →

Ca^{++} induced Ca^{++} release

N.B.: Relaxation

Ca^{++} uptake
(Ca^{++} pump)

$Na^{+}-Ca^{+}$
exchanger

Length-Tension relationship (Starling law) (MCQ)

When diastolic volume ↑

↑ Force of contraction

Within limits

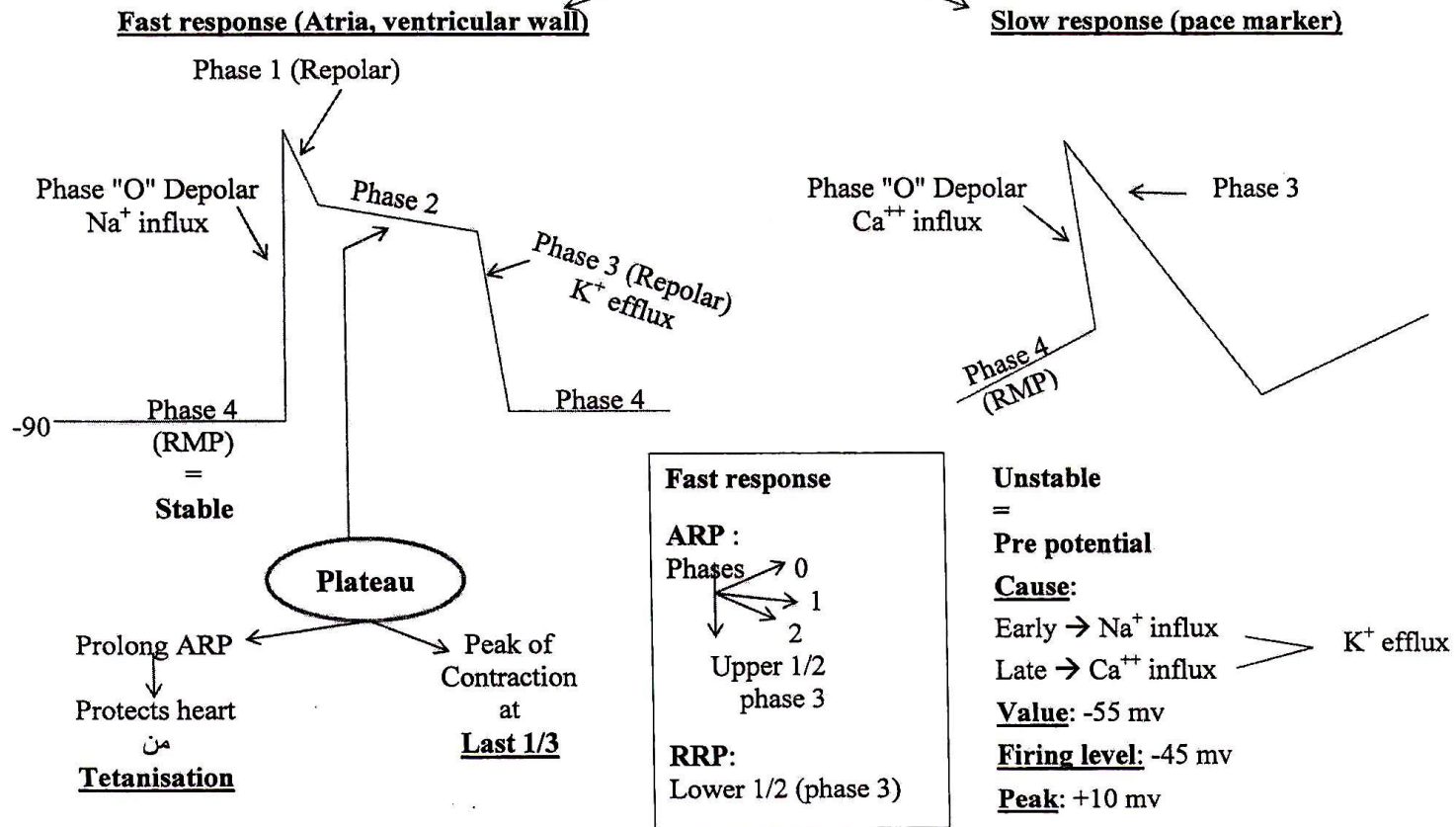
Gradation of
cardiac Ms
contraction

↑ Length

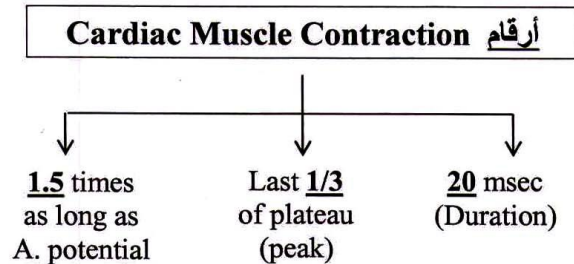
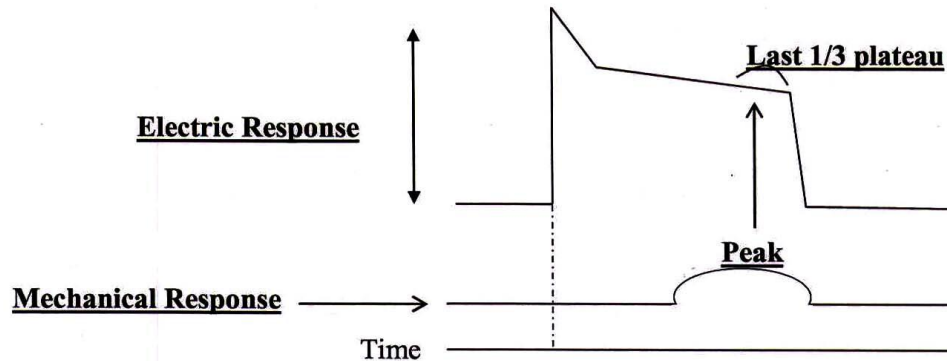
↑ Ca^{++}

No
 O_2 debt

Cardiac Muscle (Action potential) د. محمد فايز



د. محمد فايز Excitability changes of cardiac muscle

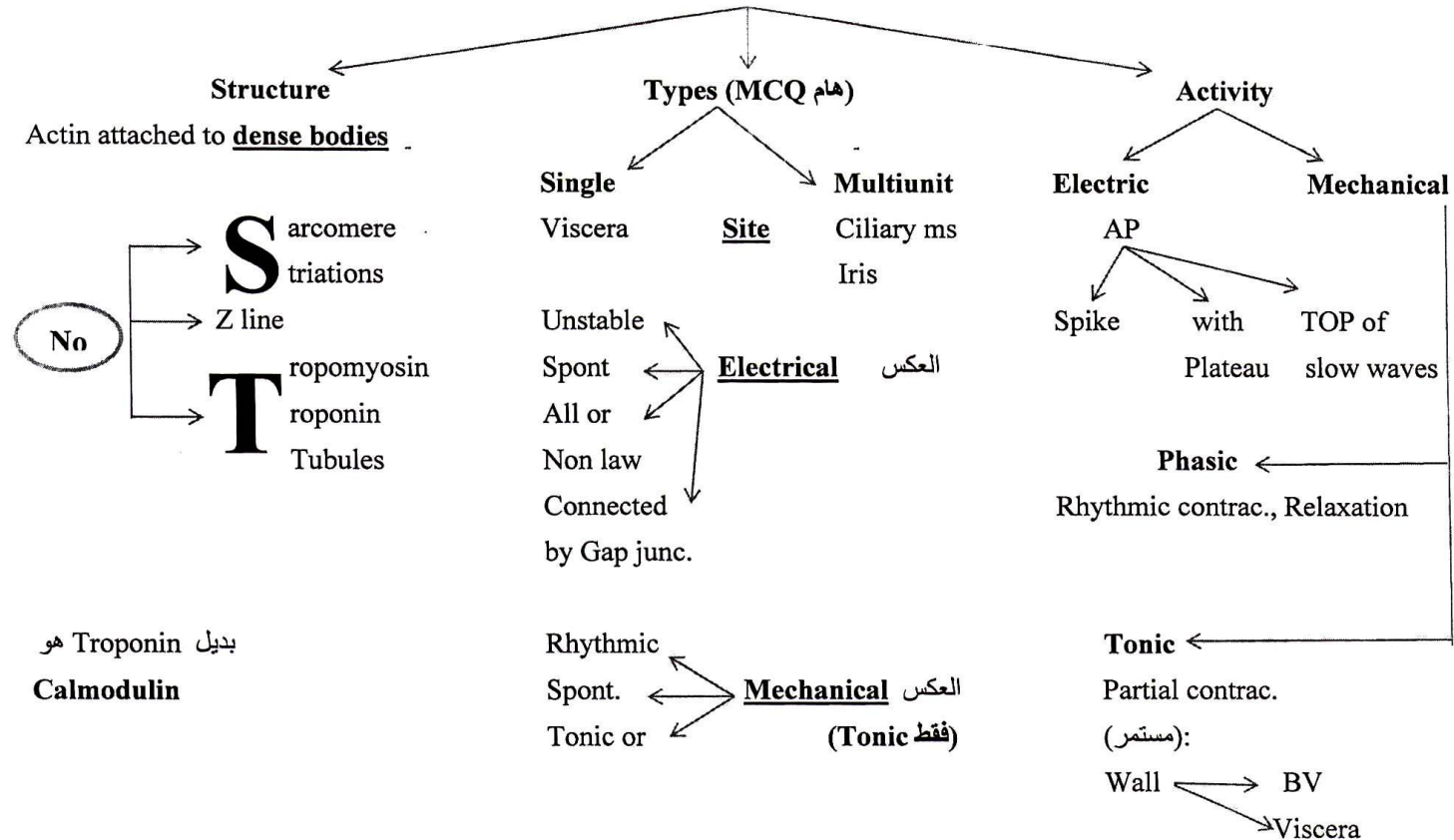


Cardiac Refractory Periods

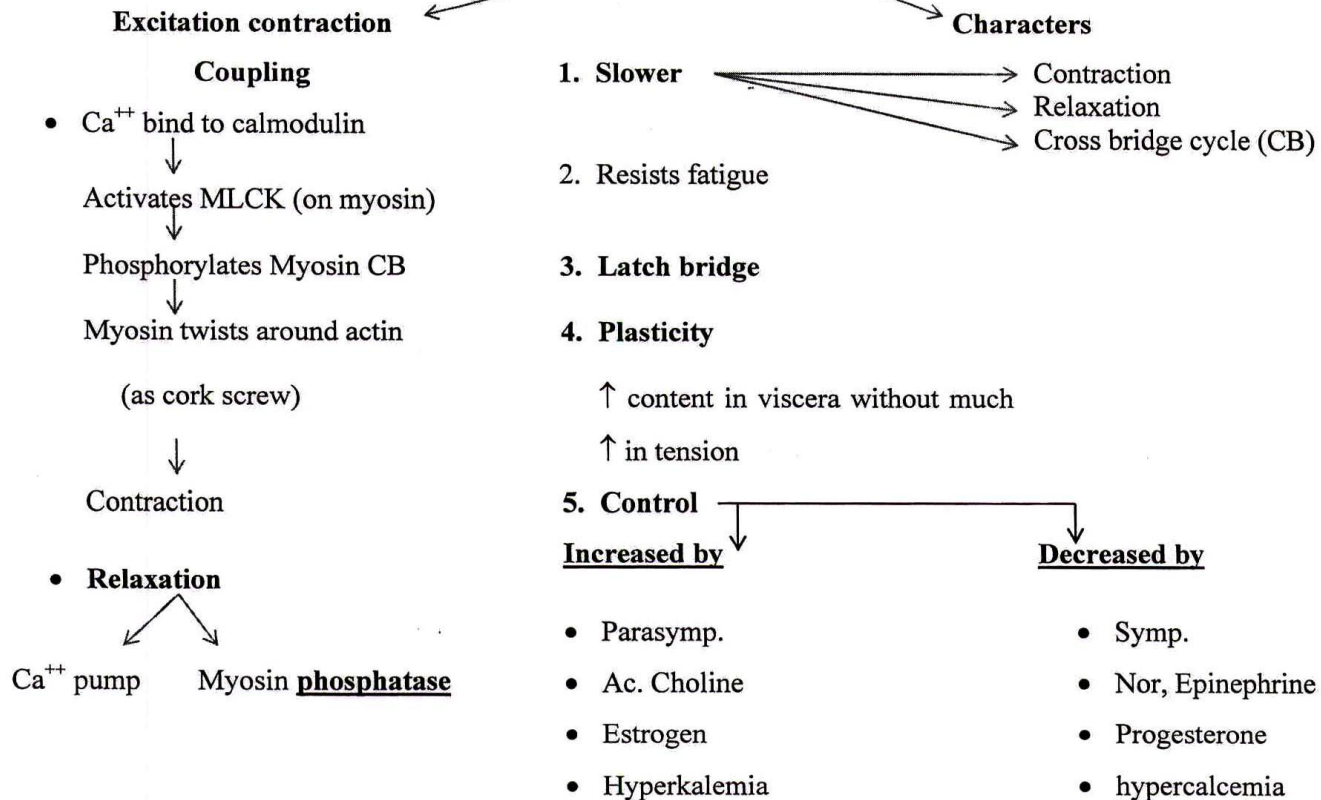
	ARP	RRP
Electric corresponds to	Phases 0, 1, 2, upper 1/2 (3)	Lower 1/2 (phase 3)
Mechanical corresponds to	* Systole * 1 st part of diastole	Middle part of diastole
Significance	Long ARP which extends to middle of diastole → Safety Vs Tetanisation	

19a

د. محمد فايز Smooth Muscle



Smooth Muscle د. محمد فايز



د. محمد فايز (10 S) Smooth Muscle



small size

striation

arcomere

لا يوجد

single unit: Gap junctions

sarcoplasmic reticulum: Poor developed

source of Ca^{++} : ECF, Ca^{++}

sensor of Ca^{++} : Calmodulin

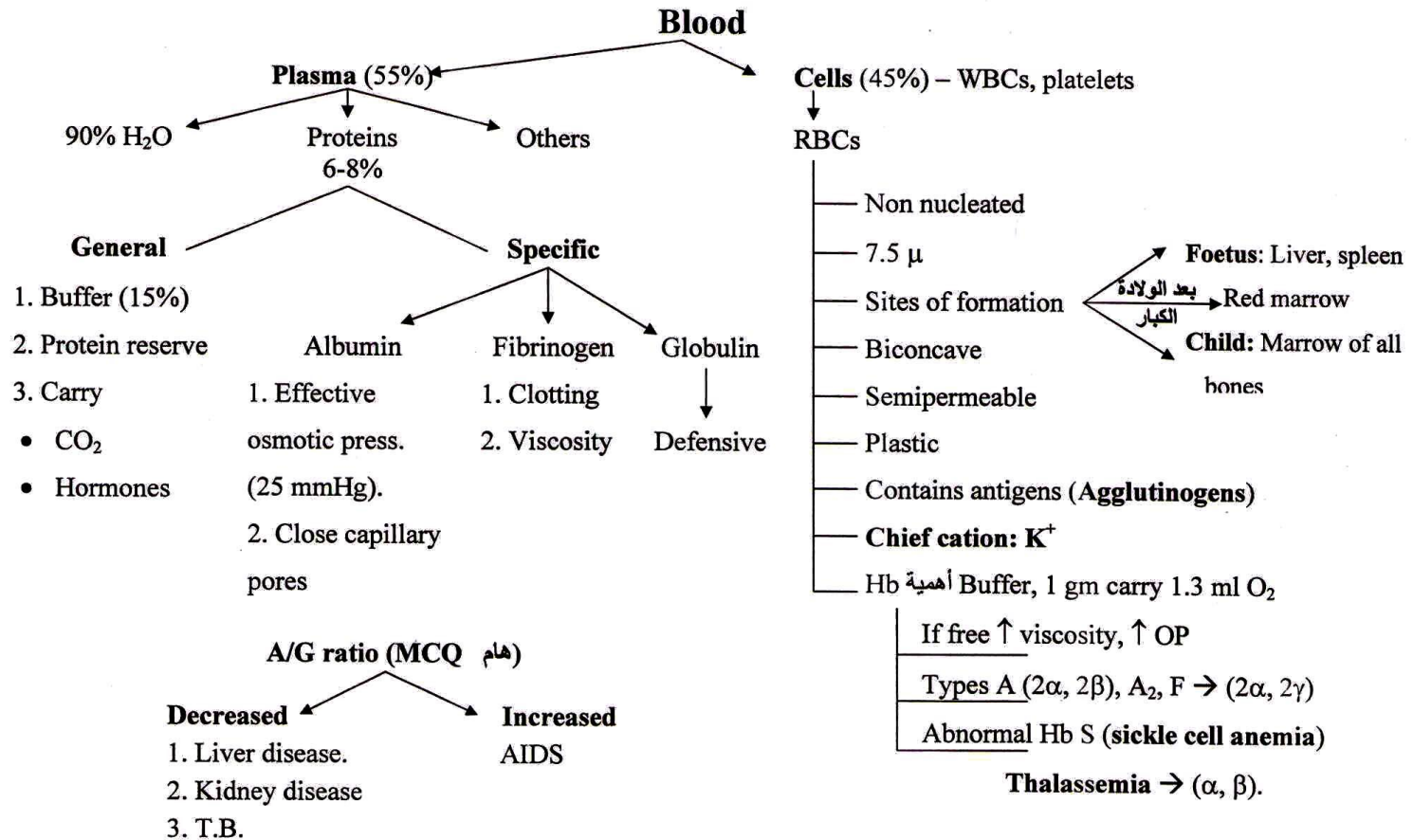
slow contraction

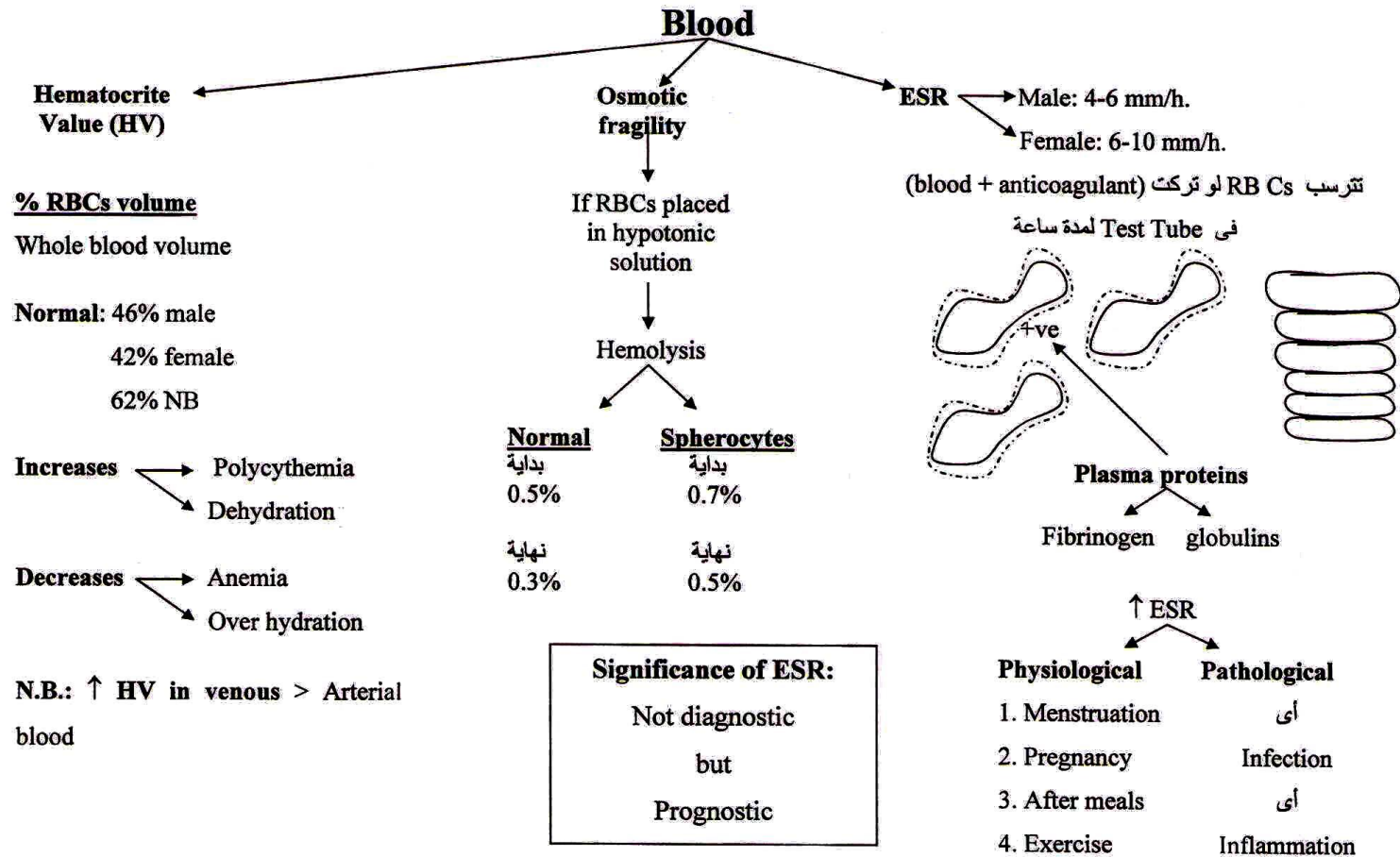
no resting potential

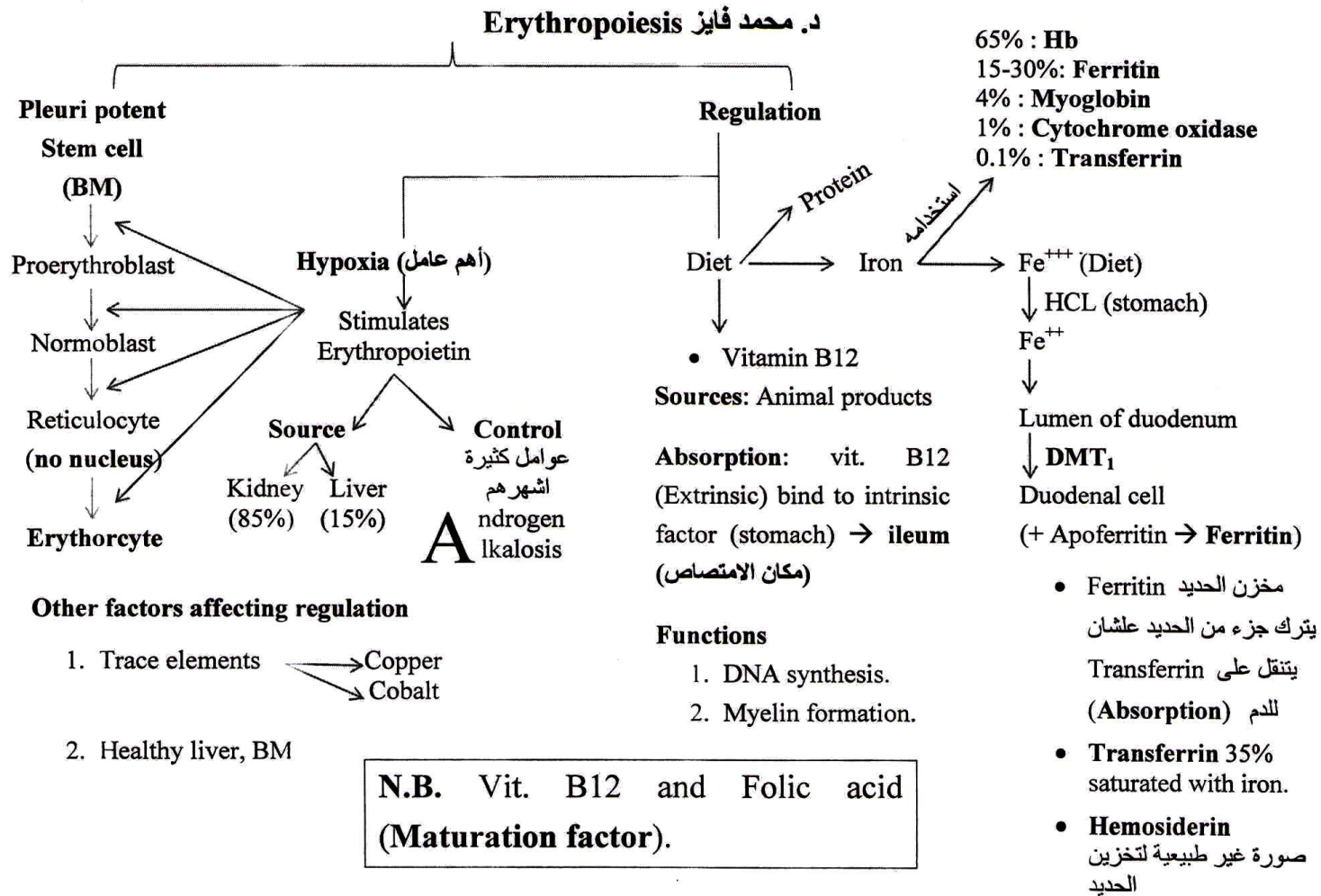
slow waves

(22)

Blood







د. محمد فايز Anemia

Detected by

(MCQ blood indices هام جدا مسائل)

(مجموعة معادلات تحدد أي نوع anemia)

$$1. \text{ MCV} = \frac{\text{HV}}{\text{RBC count}} \times 10$$

Normal: $80-95 \mu^3$

<80 → Microcytic

> 95 → Macrocytic

$$2. \text{ MCH} = \frac{\text{Hb amount}}{\text{RBC count}} \times 10$$

Normal: 27-31 pg.

<27 → Hypochromic

>31 → Hyperchromic

$$3. \text{ MCHC} = \frac{\text{Hb amount}}{\text{HV}} \times 100$$

Classification

Cell size

1. Normocytic.

2. Microcytic.

3. Macrocytic.

Cause

← **Hemorrhagic**
Acute Chronic

العيب في المصنع
(BM)
↓

Aplastic Anemia

↓
Normocytic
Normochromic

↓ Iron
↓

Microcytic Hypochromic

↓ Vit. B12
↓

Macrocytic Hyperchromic

=
Megaloblastic

=
Pernicious anemia

Others

Hemolytic

Corpusc.
Hb defect e.g.

- Hb S.
- Thalassemia.

Extra Corpusc.

Antibodies Vs RBCs

Bacterial toxins

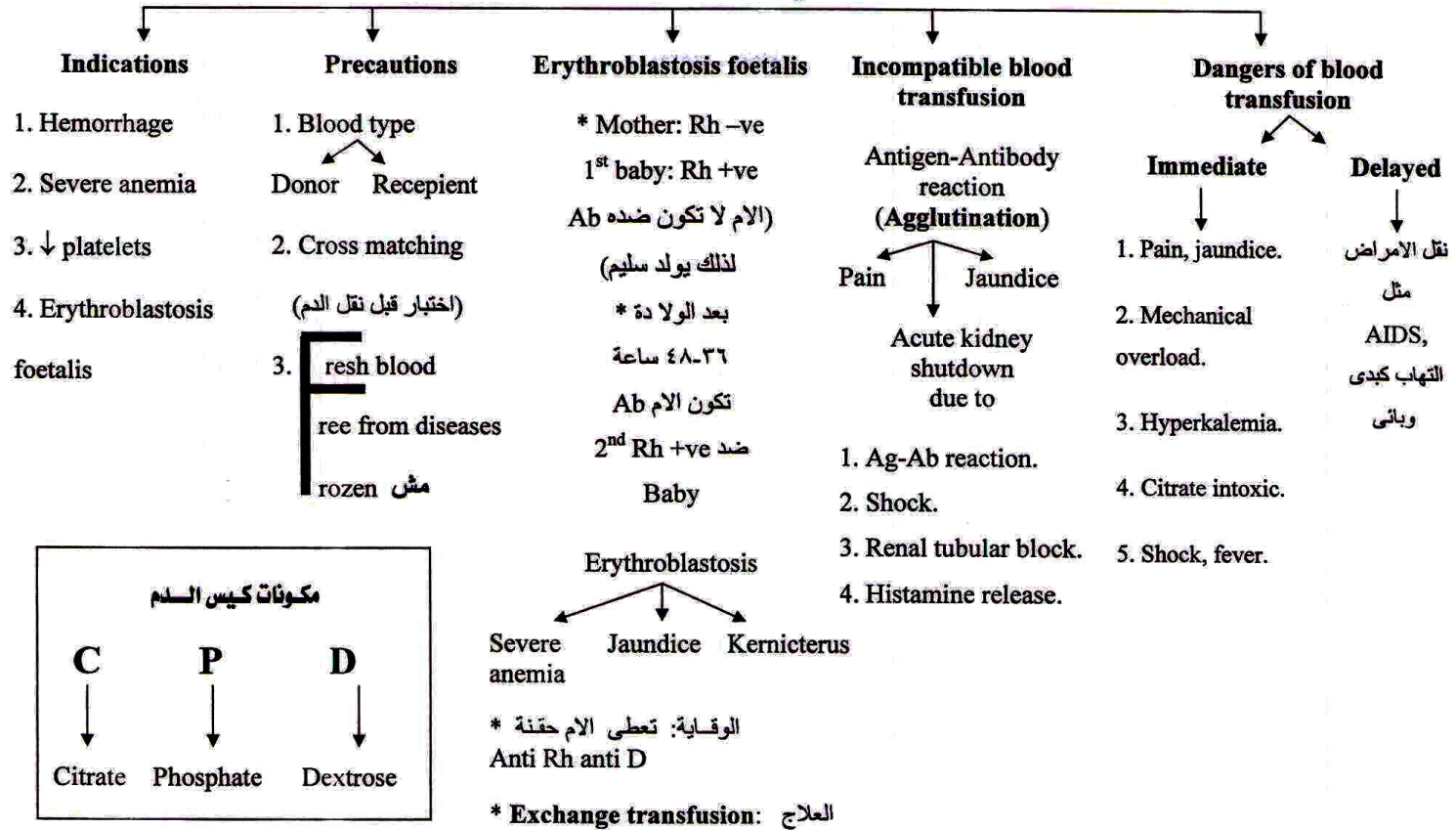
Chemicals e.g. lead

Drugs e.g. sulpha.

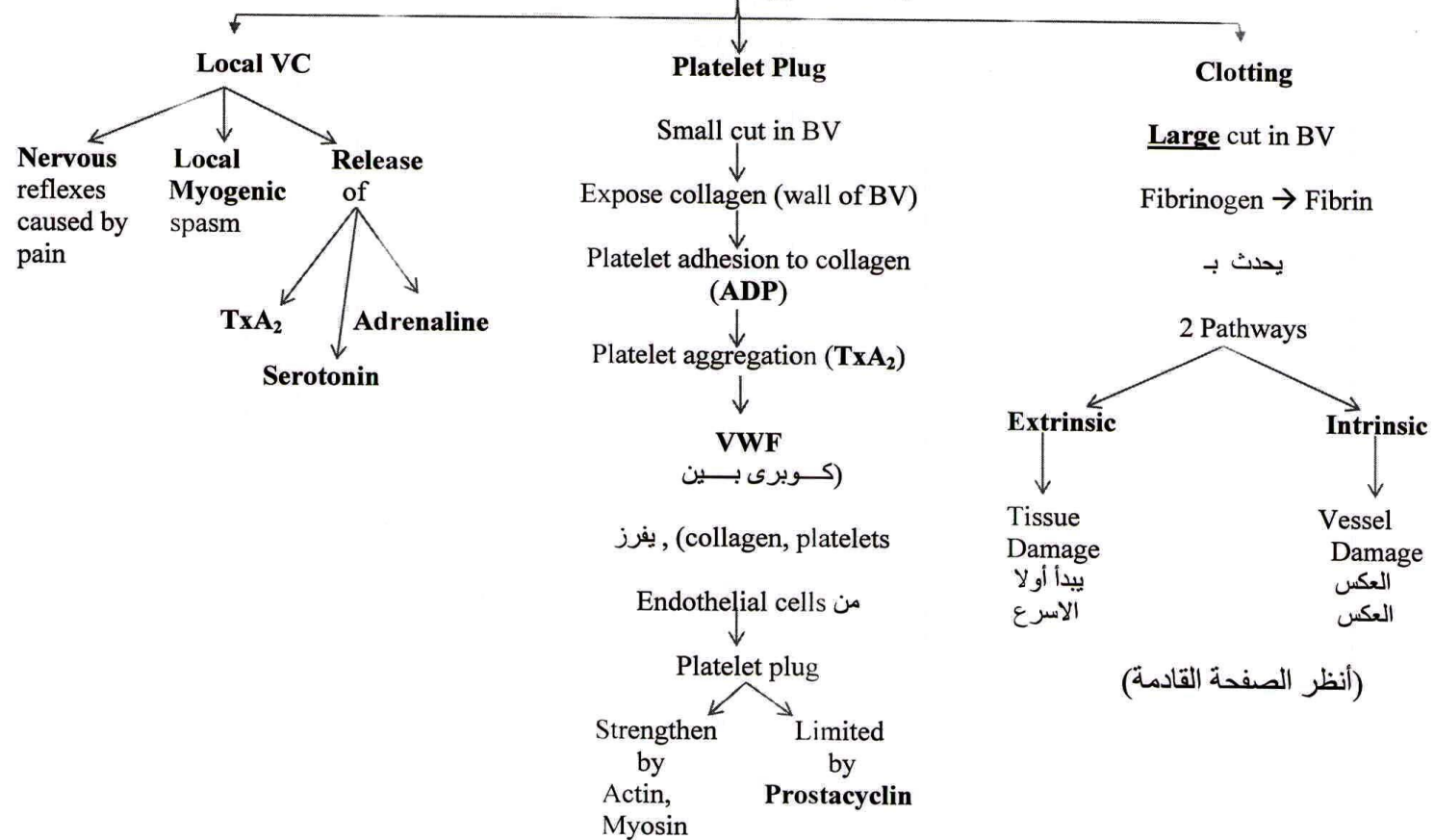
Anemia of Renal failure

↓
Normocytic
normochromic

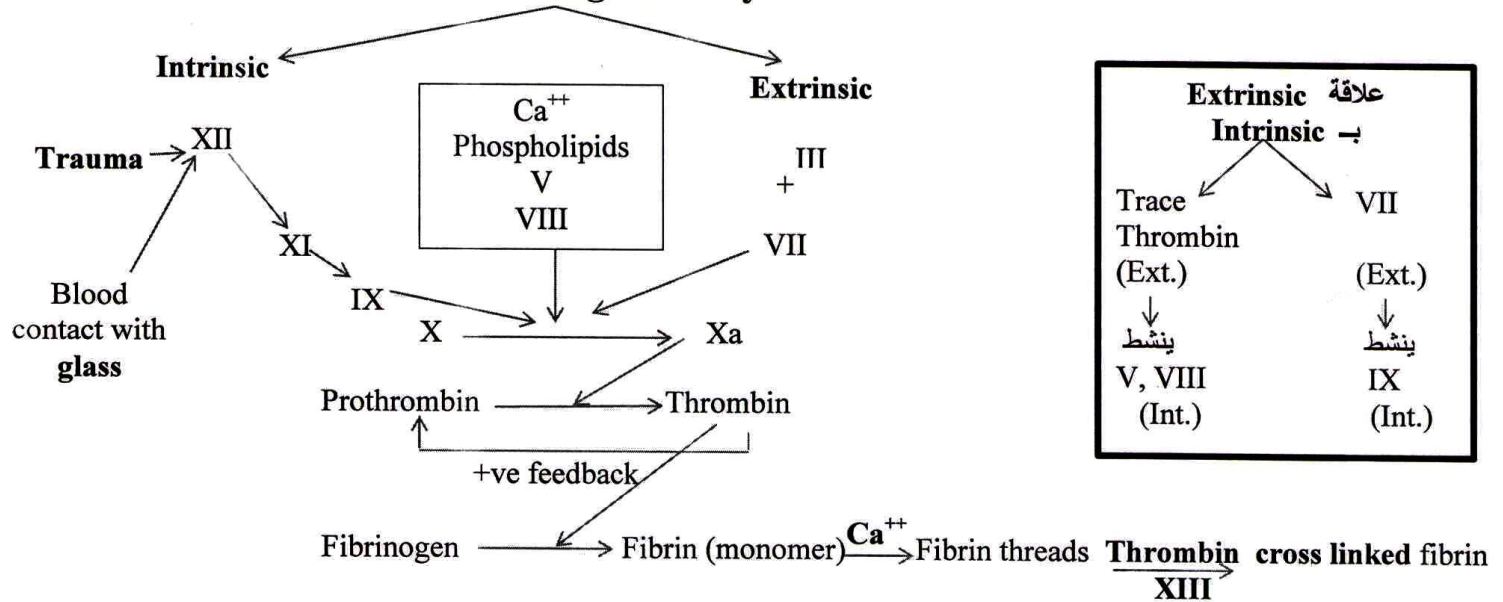
Blood Groups



د. محمد فايز Hemostasis



Clotting Pathways د. محمد فايز



ملحوظات هامة

Platelets دور

1. Bond fibrin threads
2. Release XIII
3. Activate contractile proteins

Clot retraction

Clot contracts
↓
Pull edges of Broken vessels

Intestinal flora Source

Source Absorption Bile

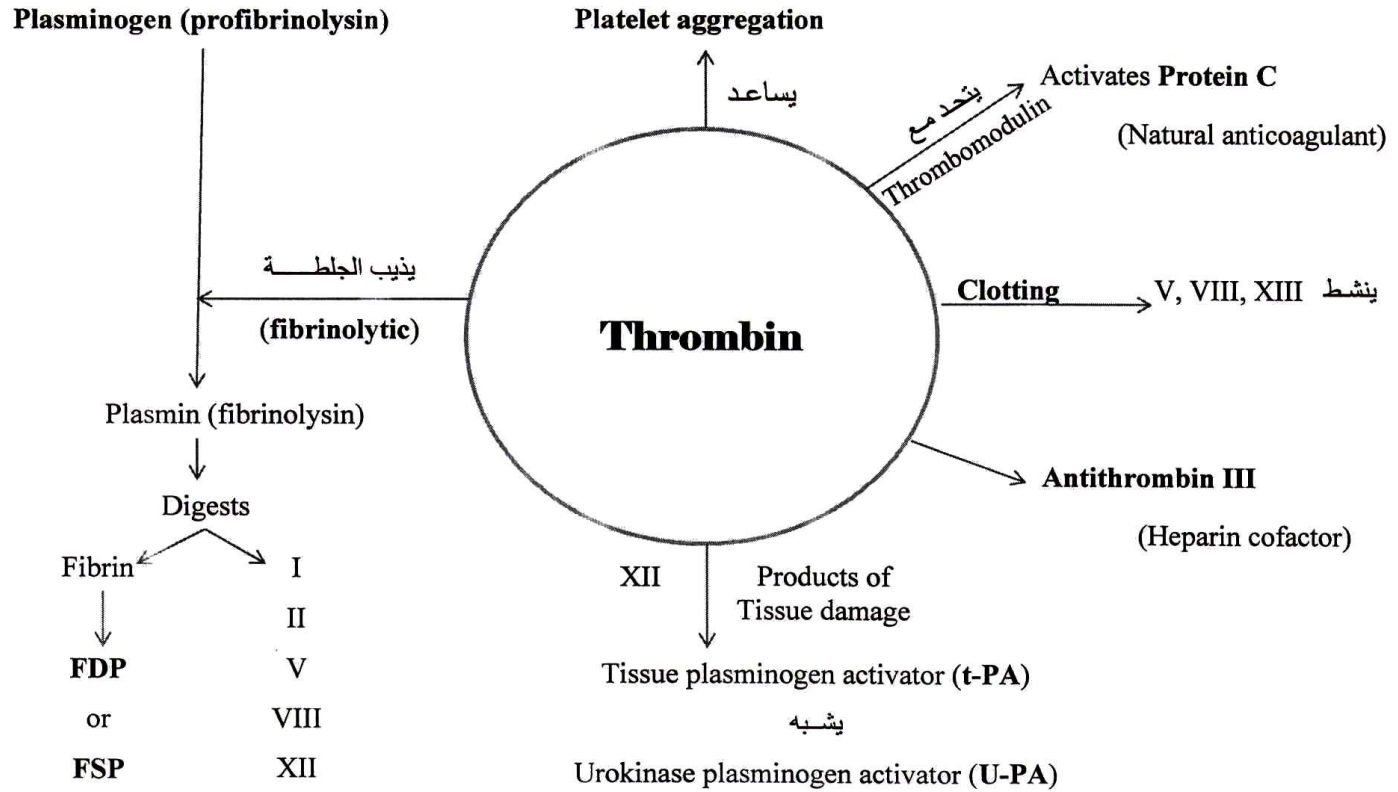
Vitamin K

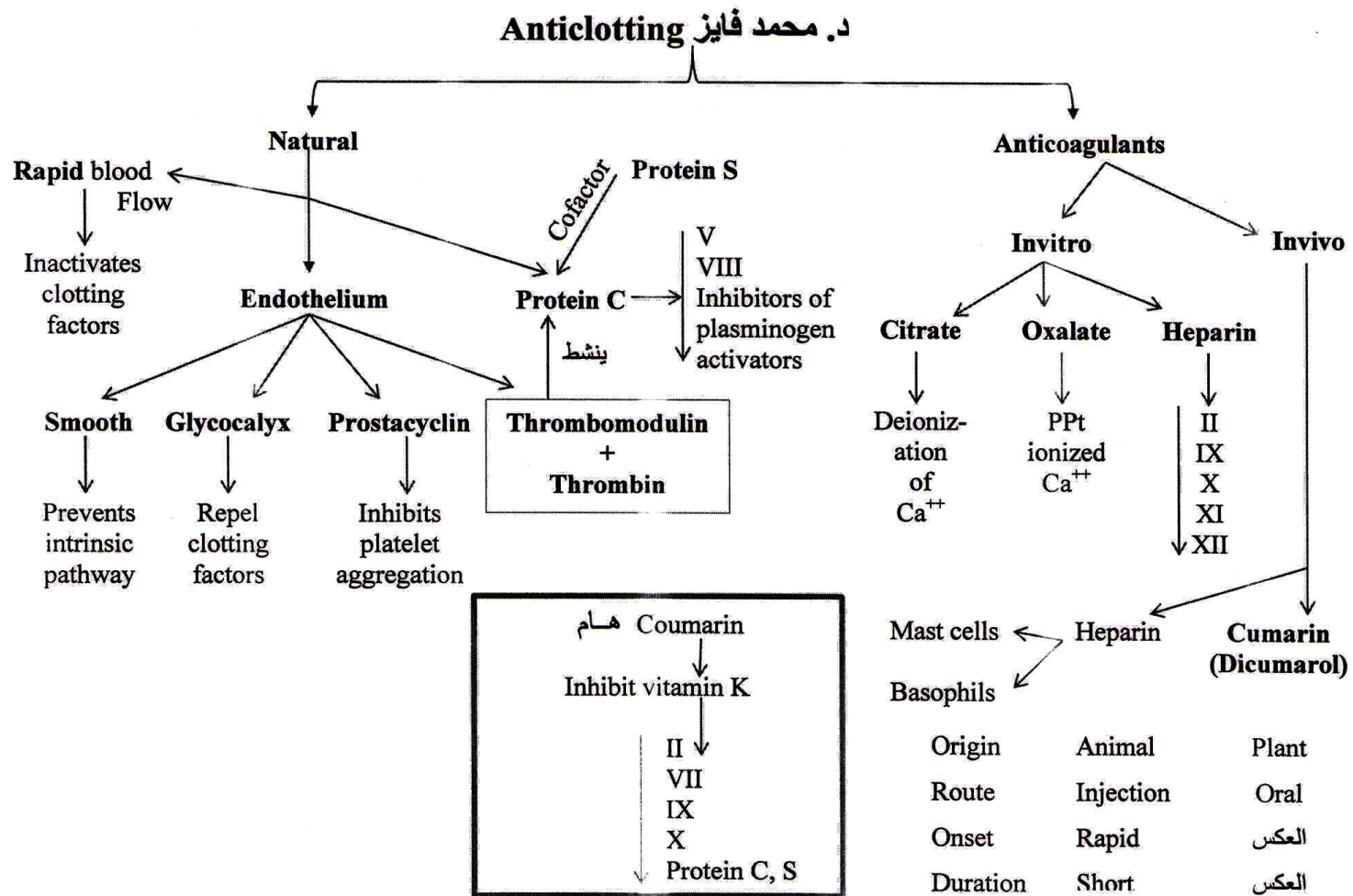
- Fat soluble needs Bile for absorption
- Synthesize II, VII, IX, X

Ca⁺⁺ هام

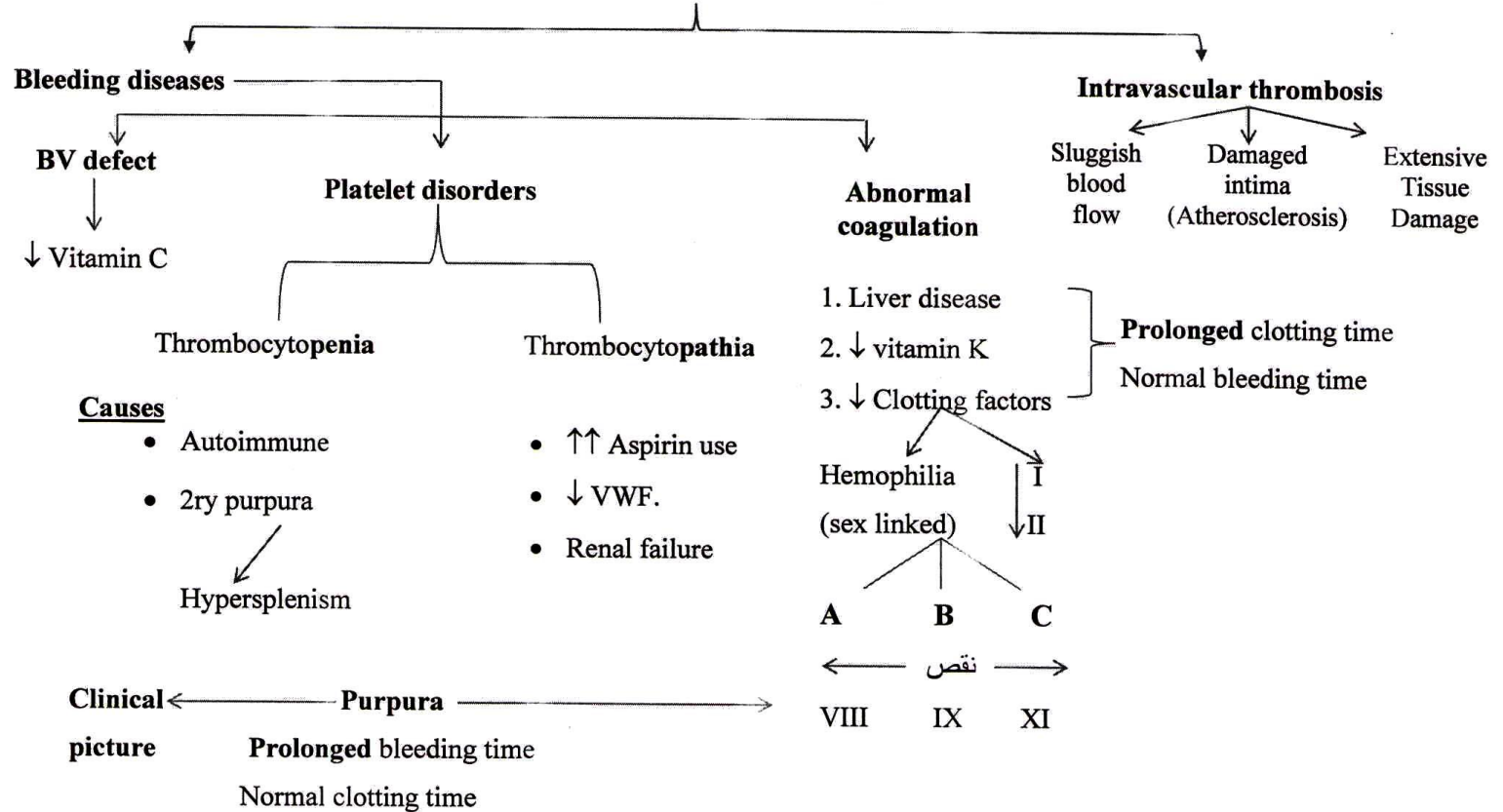
لكل الخطوات
ماعدًا اول اثنين
Intrinsic

د. محمد فايز Thrombin

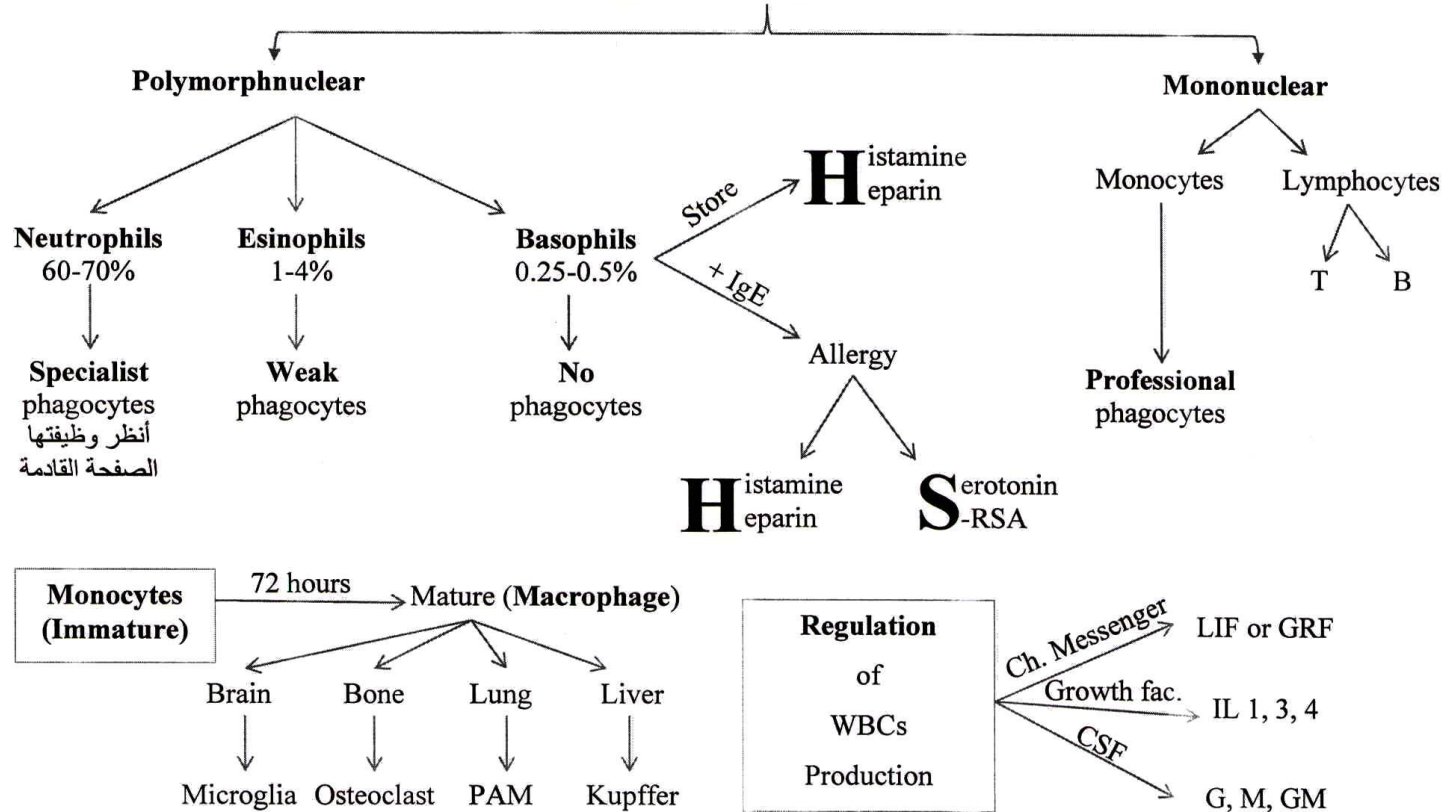




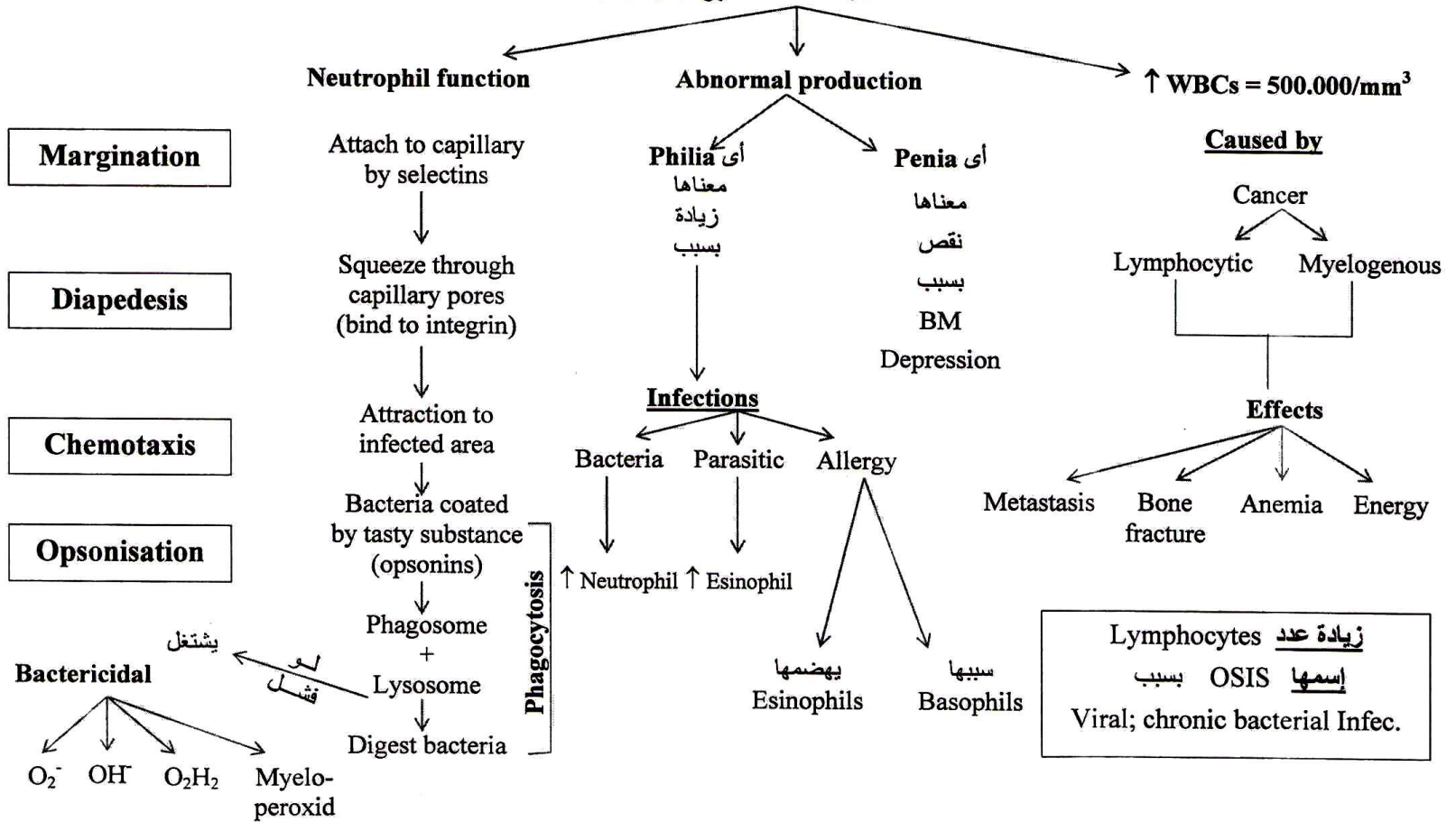
Abnormalities of Hemostasis د. محمد فايز



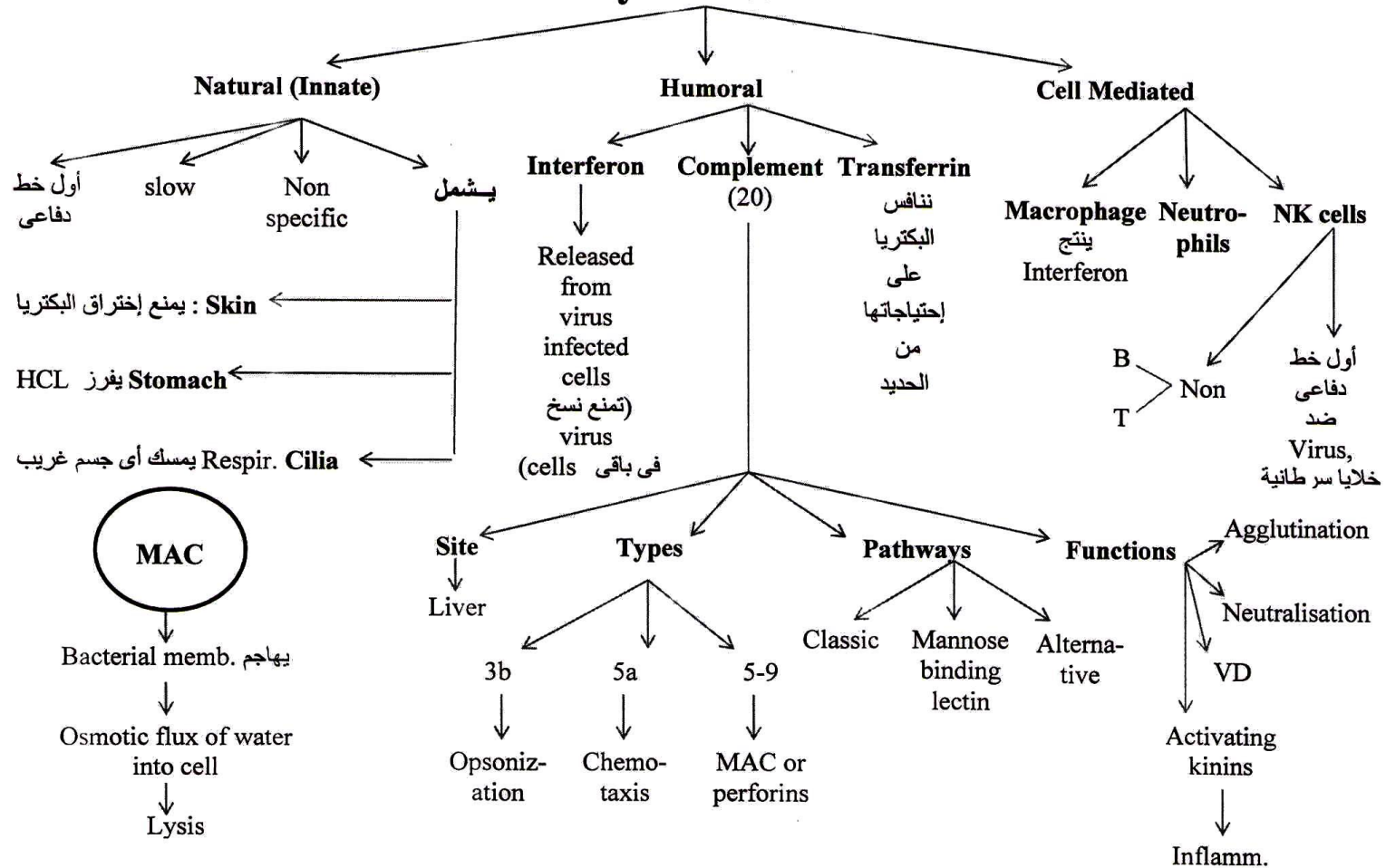
د. محمد فايز WBCs



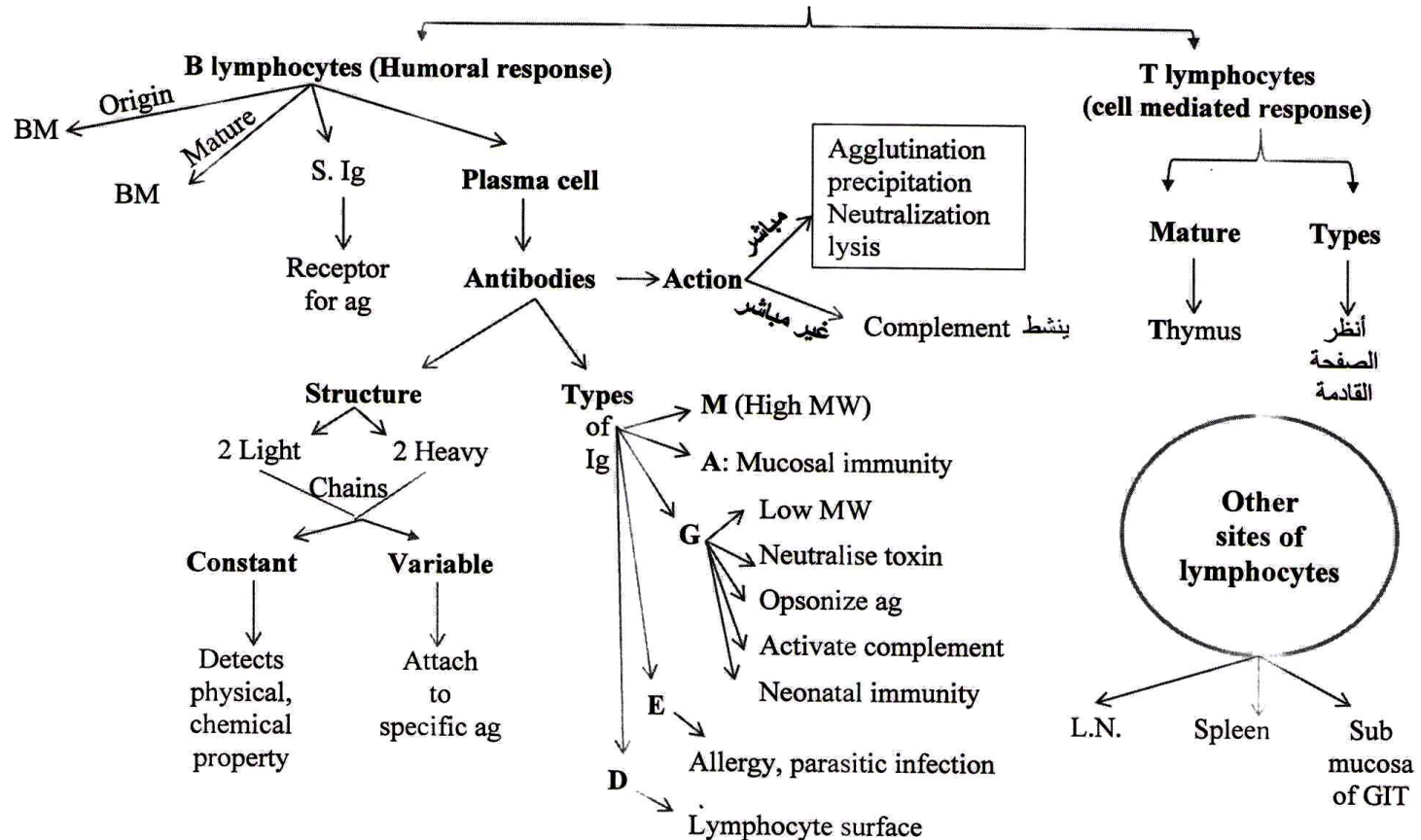
د. محمد فايز WBCs



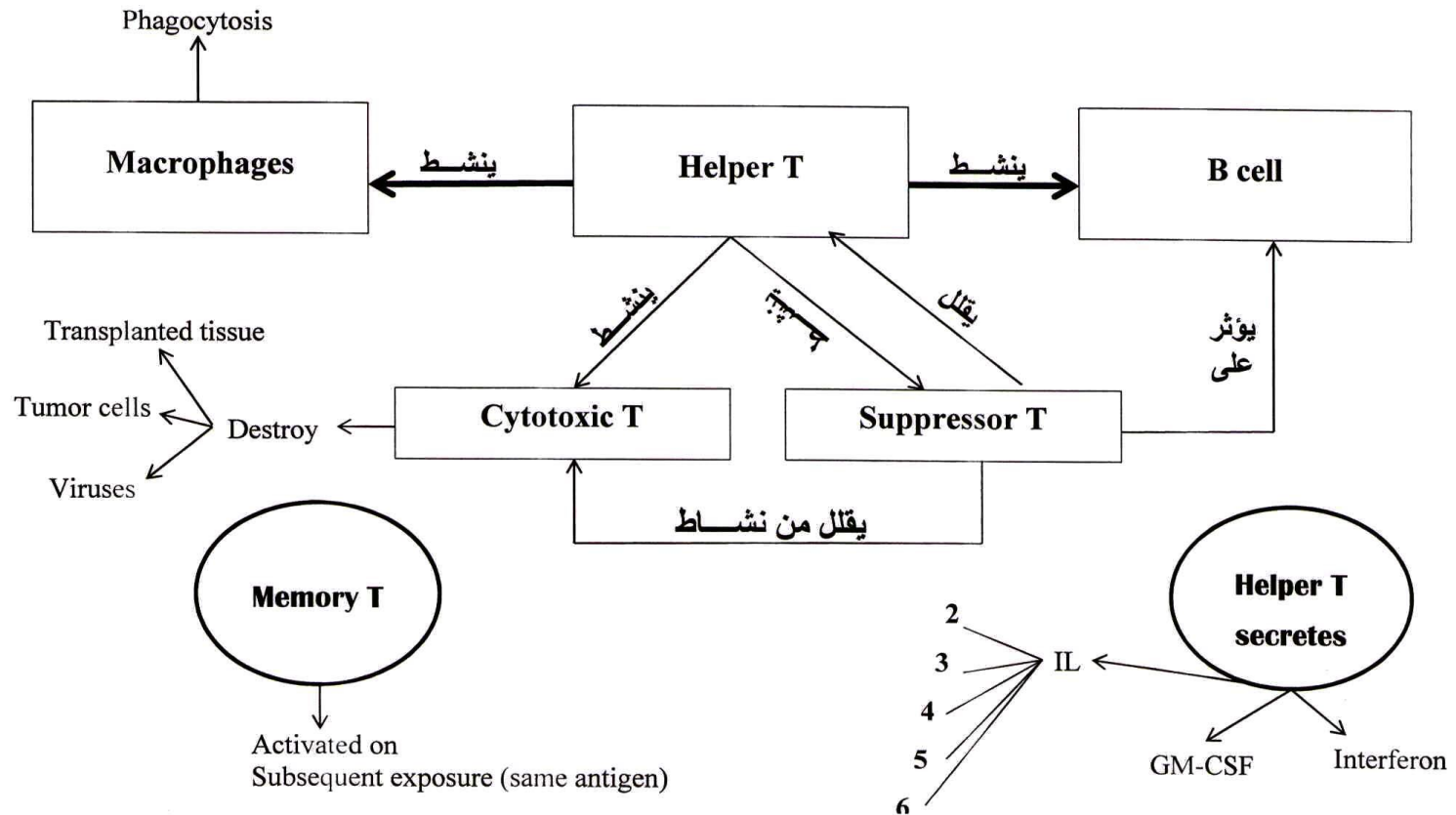
د. محمد فايز Immune System



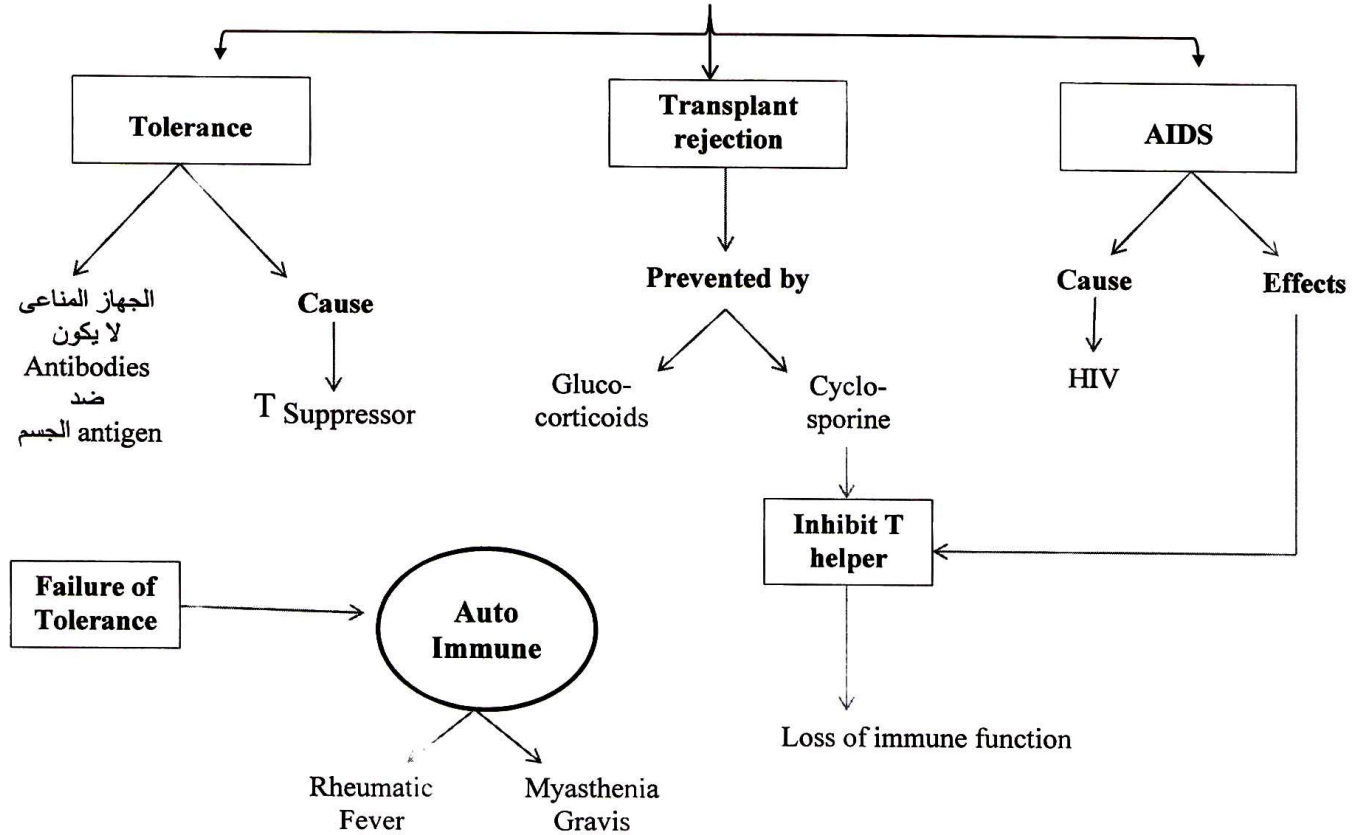
د. محمد فايز Acquired Immune System



د. محمد فايز Types of T cells

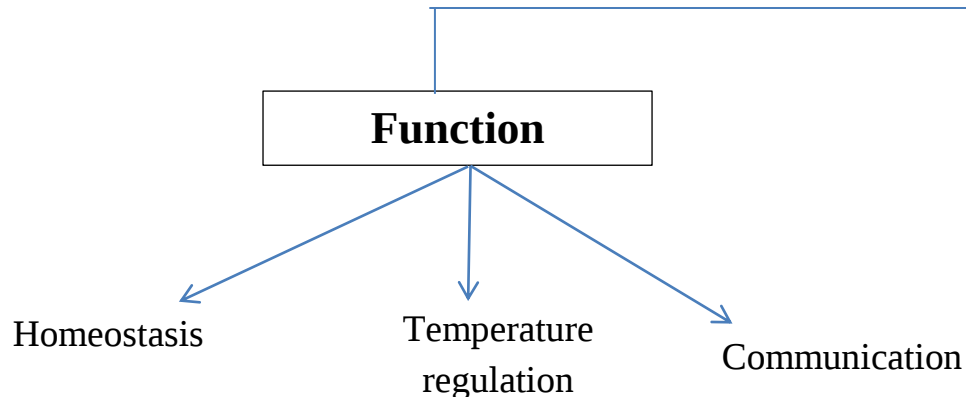


د. محمد فايز Notes



Cardiac

د/ محمد فايز CVS



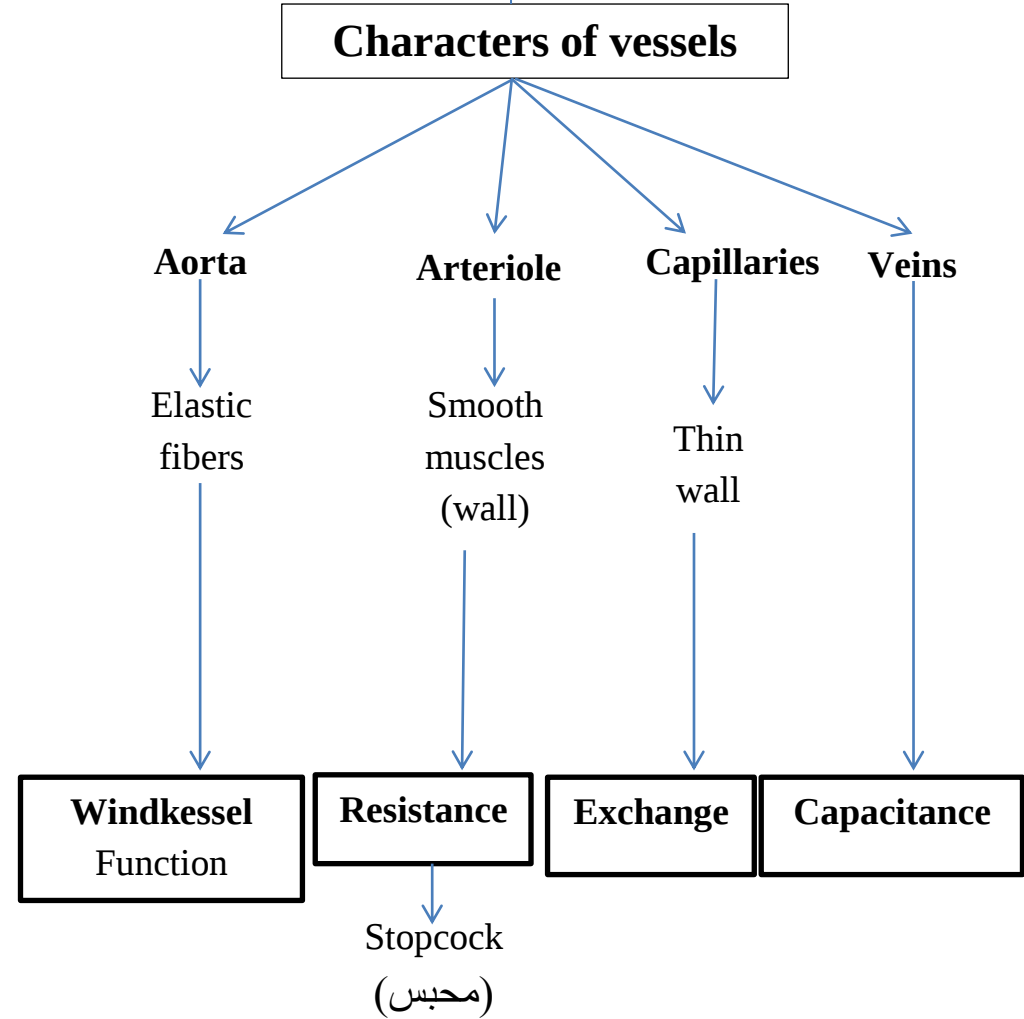
Capacitance

$$C = \frac{\Delta V}{\Delta P}$$

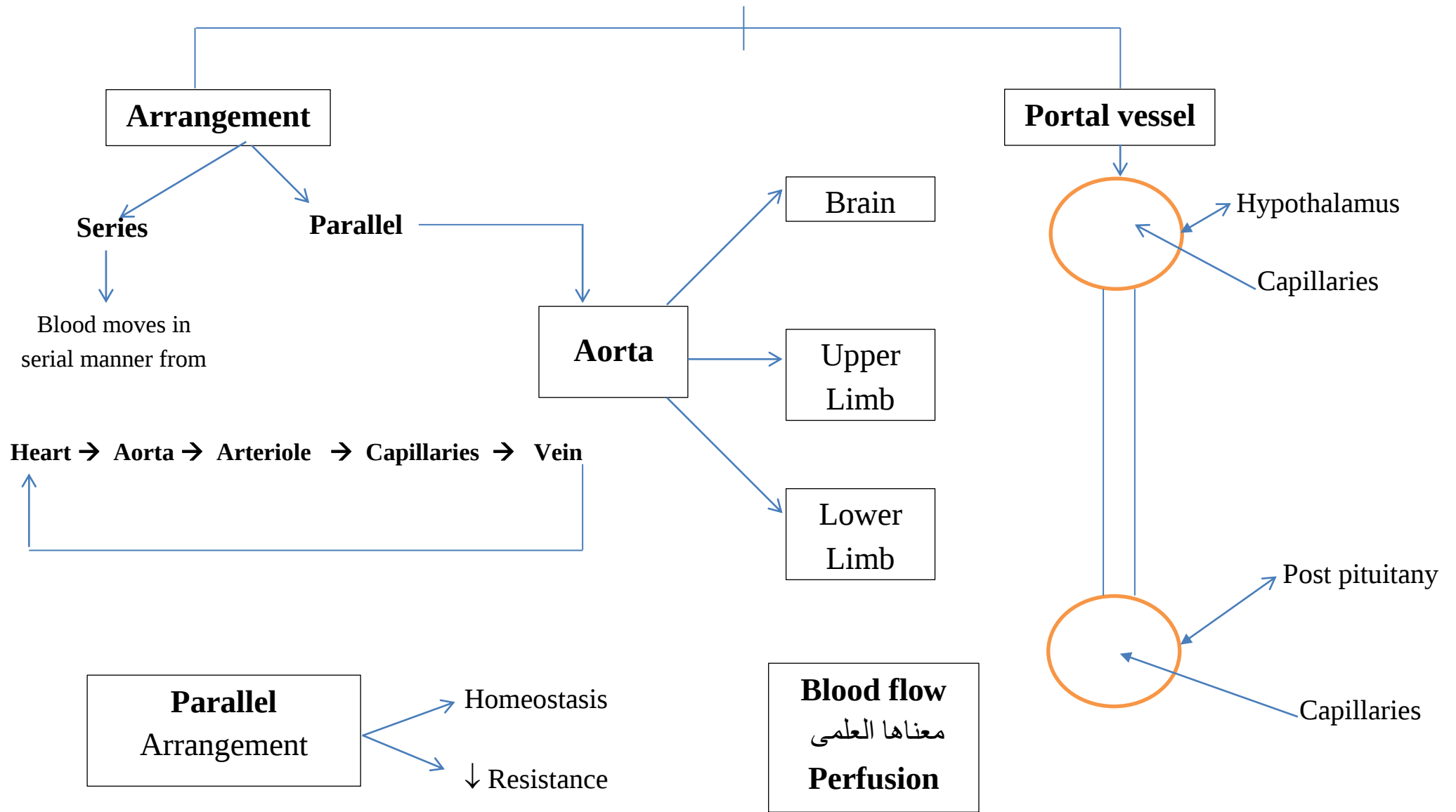
المعنى:
يمتلئ باكثر حجم دم
باقدر ضغط ممكن

الدليل : 54% blood (vein)

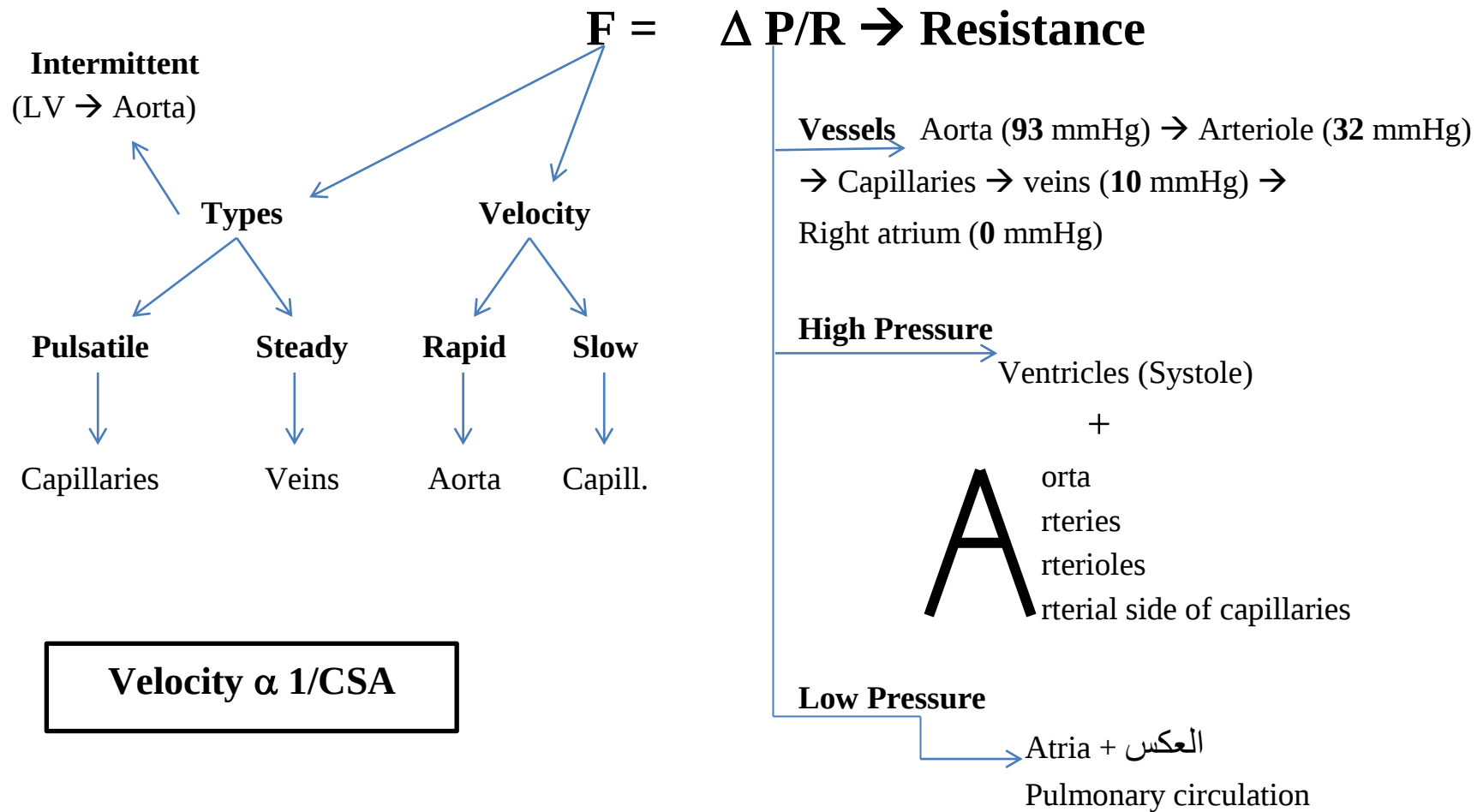
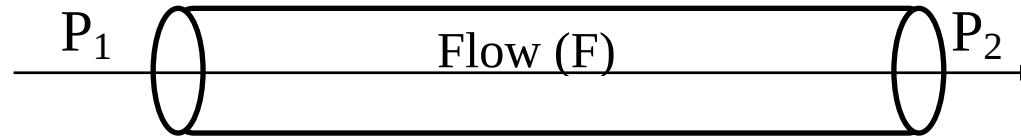
MCQ



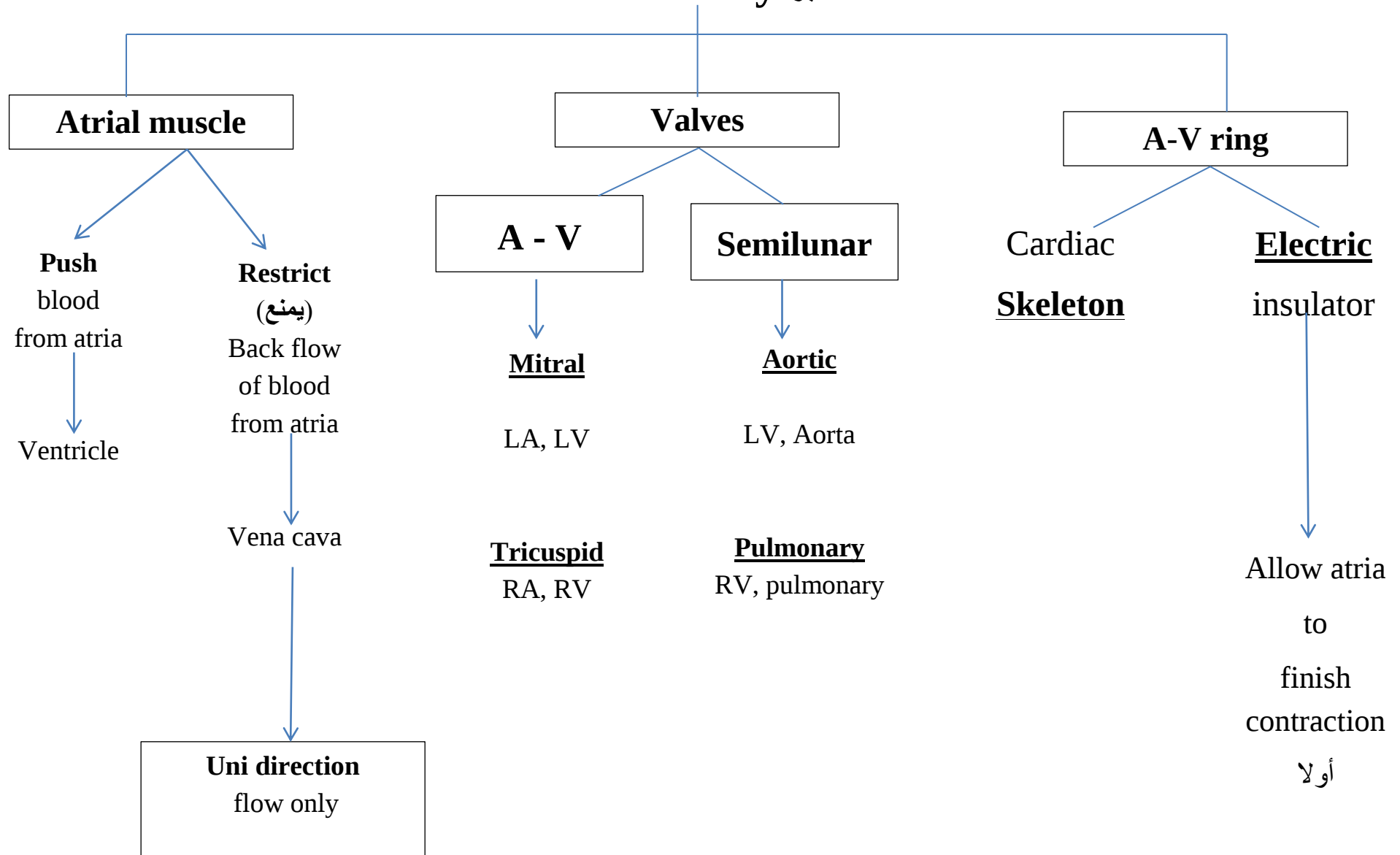
د. محمد فايز CVS



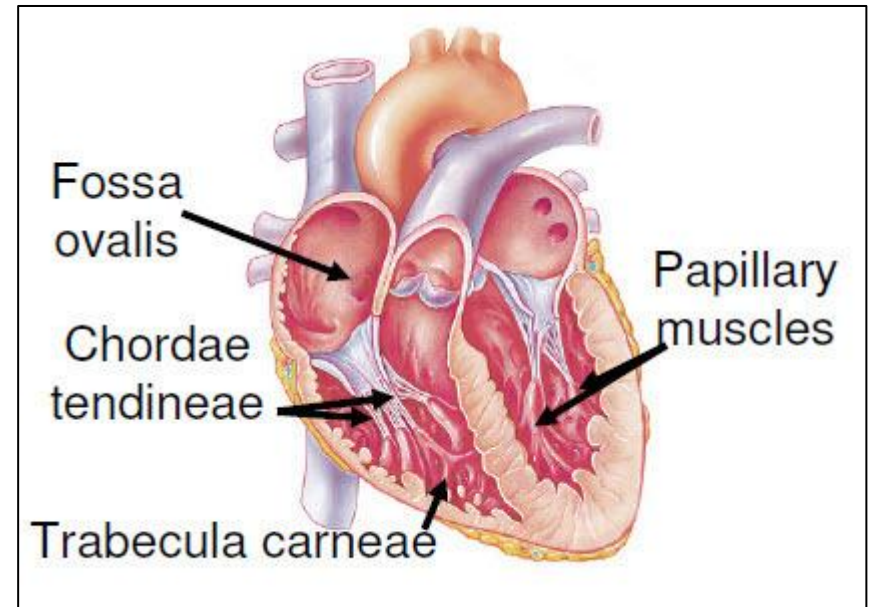
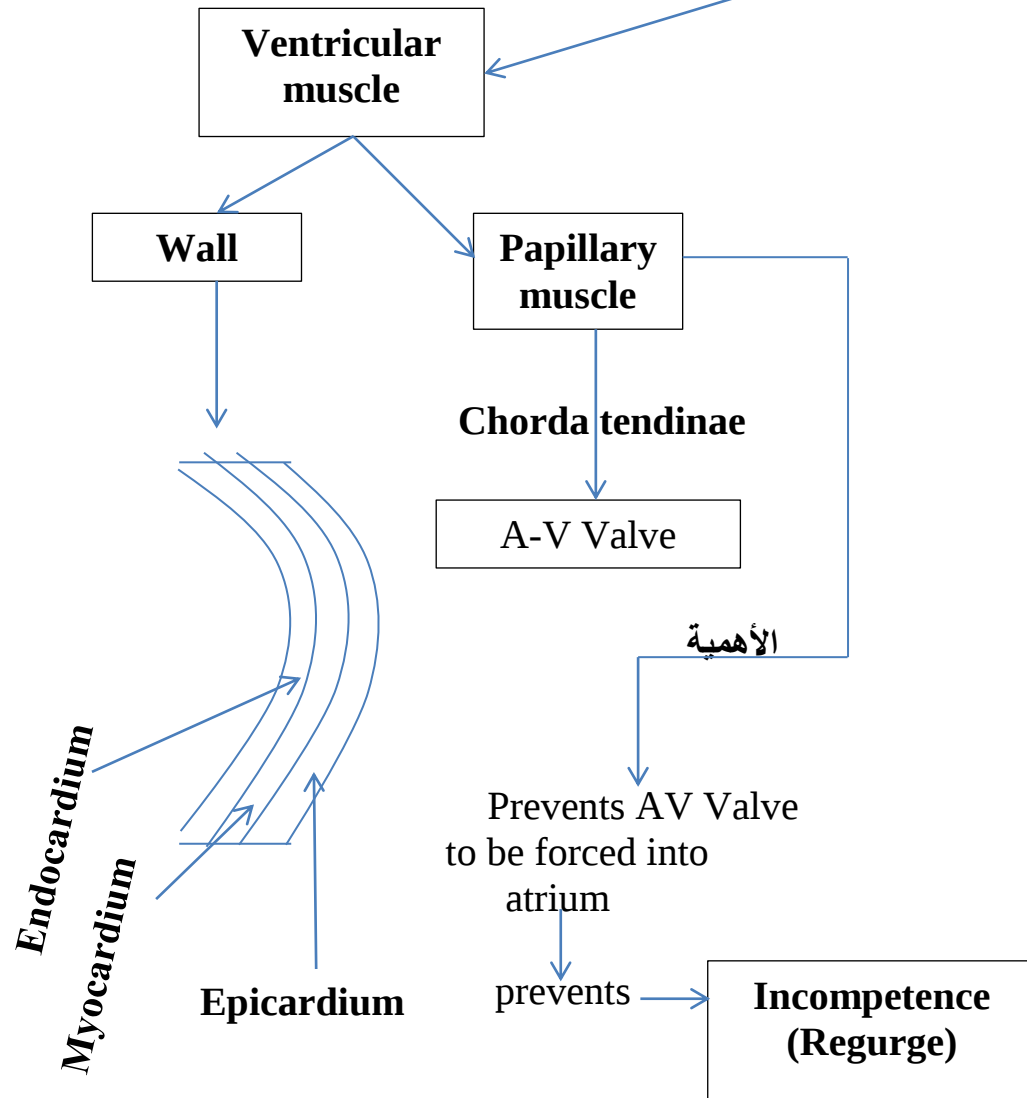
د. محمد فايز CVS



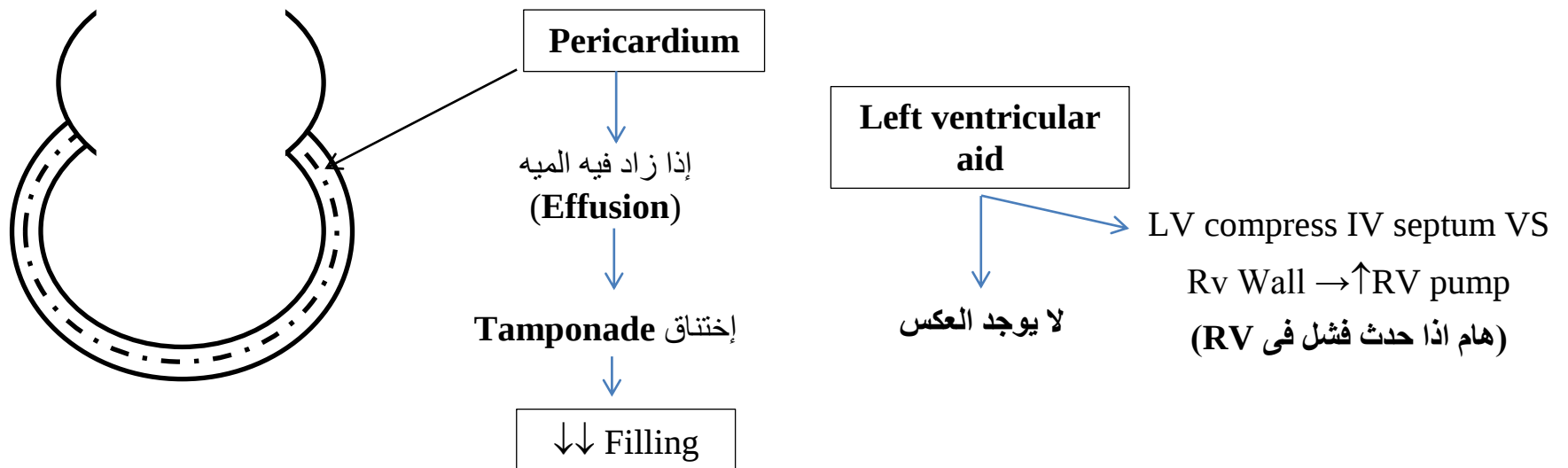
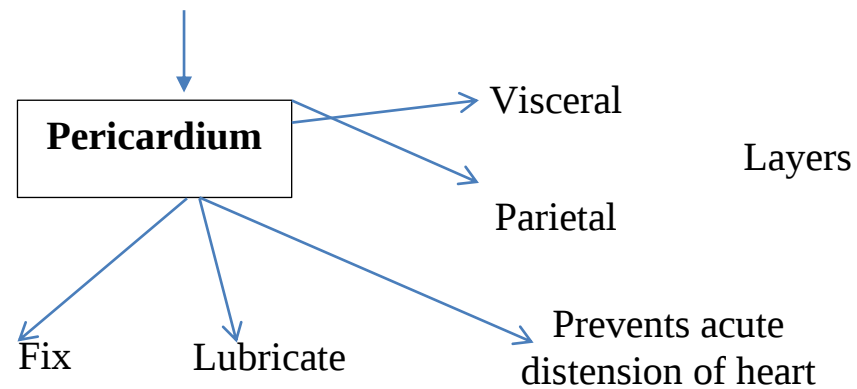
Cardiac Anatomy د/محمد فايز



Cardiac Anatomy د/ محمد فايز



Cardiac Anatomy د/ محمد فايز



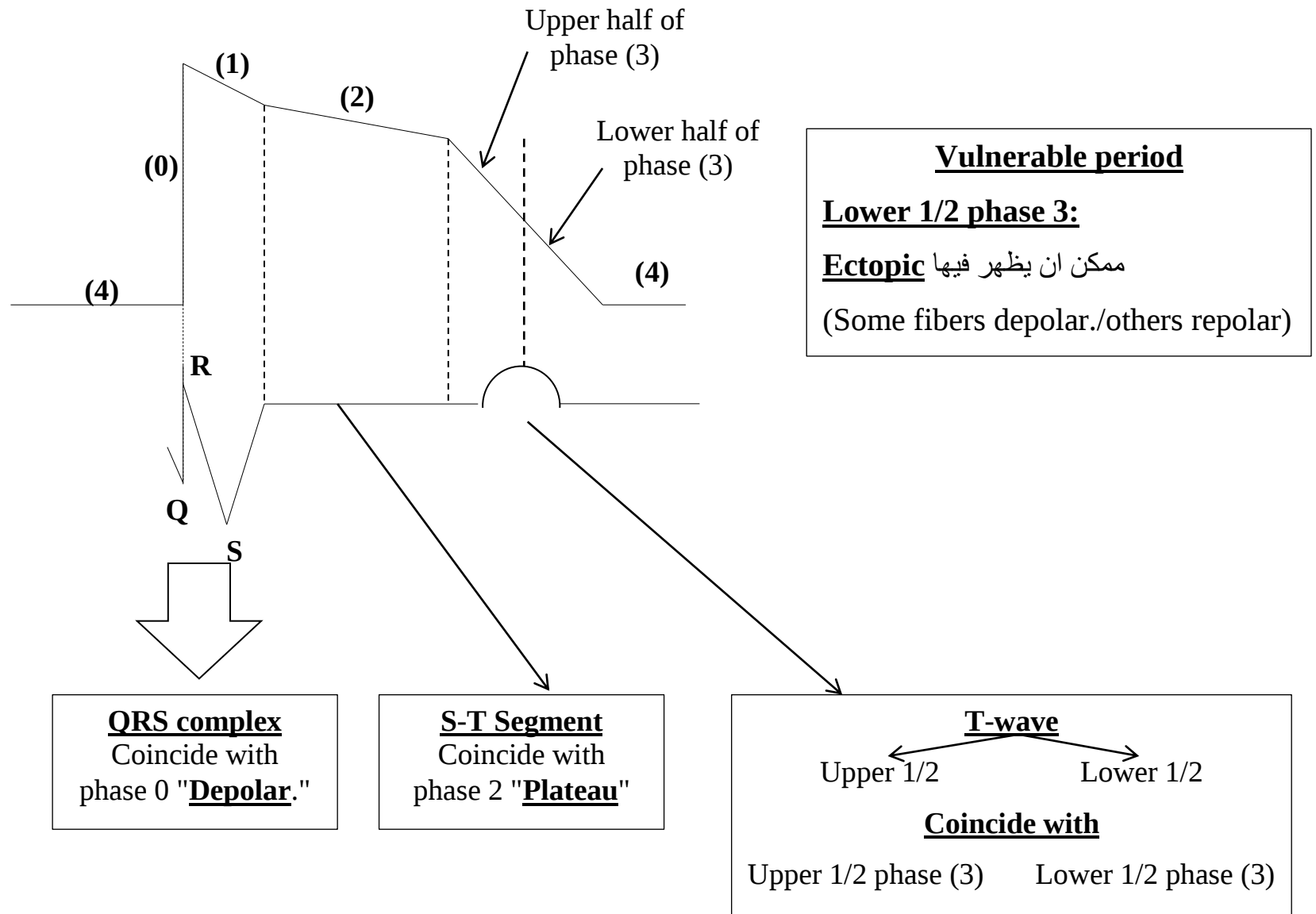
	Fast Response	Slow response
<u>Phase 4</u> Cause Value	<u>Stable</u> ← RMP K^+ efflux > Na^+ influx -90 mV	→ <u>Unstable</u> (Prepotential) = Diastolic depolarization <u>Early:</u> Na^+ influx <u>Late:</u> Ca^{++} influx (T-channels) - 55 mV
<u>Phase 0</u> Cause Firing level	← Depolarisation Na^+ influx (voltage gated Na^+ channels) - 70 mV (Peak: +30 mV)	→ Ca^{++} influx (L-type Ca^{++} channels) - 45 mV (Peak: +10 mV)
<u>Phase 1</u>	Open voltage gated k^+ channels	-----
<u>Phase 2</u> <u>(platau)</u>	Early: ca^{++} Influx = K^+ efflux Late: Na^+ influx = K^+ efflux	-----
<u>Phase 3</u>	Open voltage gated k^+ channels	

Repolarisation

Refractory Periods دكتور / محمد فايز

	Fast Response	Slow response
Refractoriness	Shorter	Longer → Start phase 4 (post repolar. Refractoriness)
<u>ARP</u> Time Significance	Phases (0, 1, 2, upper 1/2 (3)) Safety against tetanisation	Phase (0) → 2/3 phase (3) Voltage dependent refractoriness
<u>ARP</u> Time Significance	Lower 1/2 (phase 3) Partial recovery of fast Na ⁺ channels	Late 1/3 phase (3) → start phase (4) Pathological importance of AVN (2)
<u>ERP</u> Time Significance	ARP + 1 st 10 mv of RRP → 60 mv As ARP + cardioversion (الهدف الغانه حتى يرجع SAN لطبيعته) (2000-3000 volt) in V.F	_____
<u>Supernormal period</u> Time Significance		Short at end of phase (3), start phase (4) Propagated AP → ↑ excitability

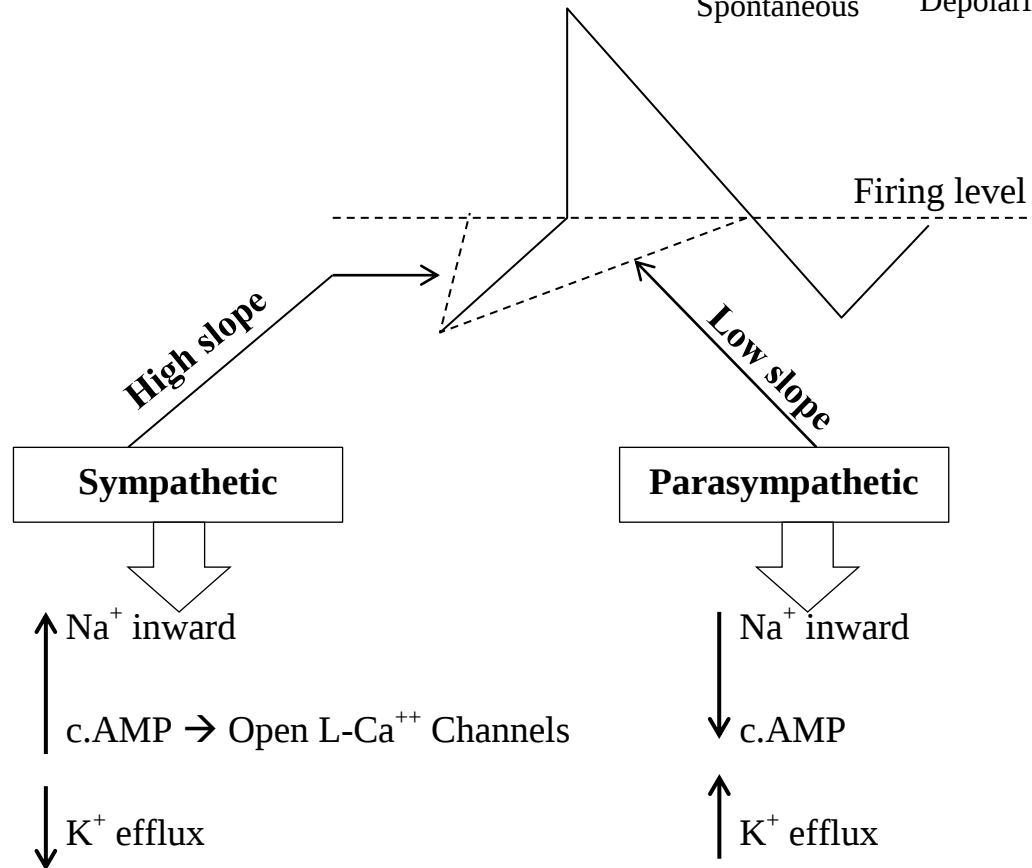
د. محمد فايز Relation between Fast response, ECG



د. محمد فايز SAN

RMP = Phase 4 = Prepotential = S S D D

Slow
Spontaneous
Diastolic
Depolarization

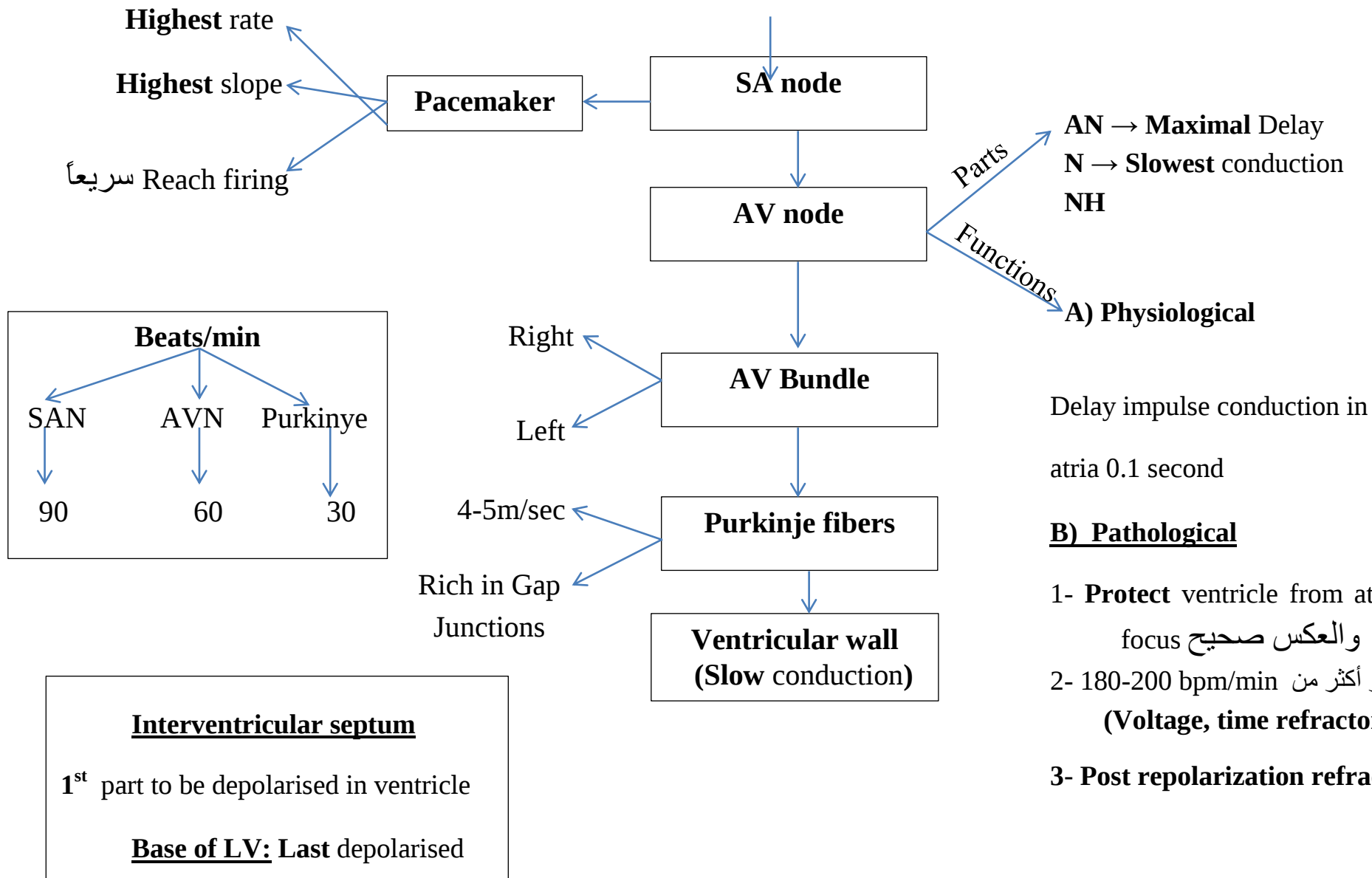


SAN is Normal Pacemaker

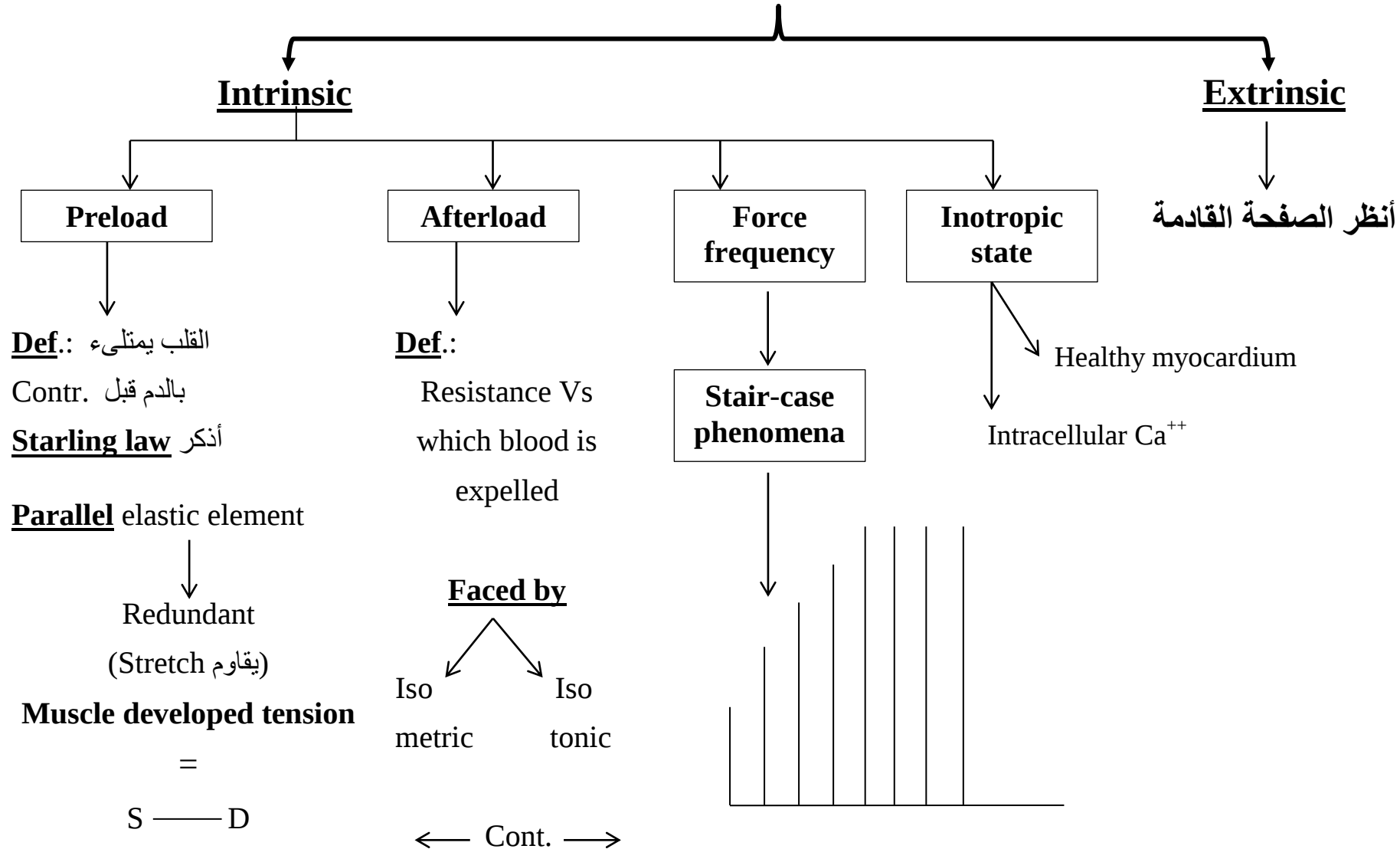
Highest → Slope
→ Discharge
→ Repolarized
Low Amplitude

	↑ SAN firing	↓ SAN firing
Nervous	Sympathetic	Parasymp.
Ions	Hypocalcemia	Hyperkalemia calcemia
Drug	A drenaline tropine	Muscarine β -Blockers
Temperature	Warming	Cooling

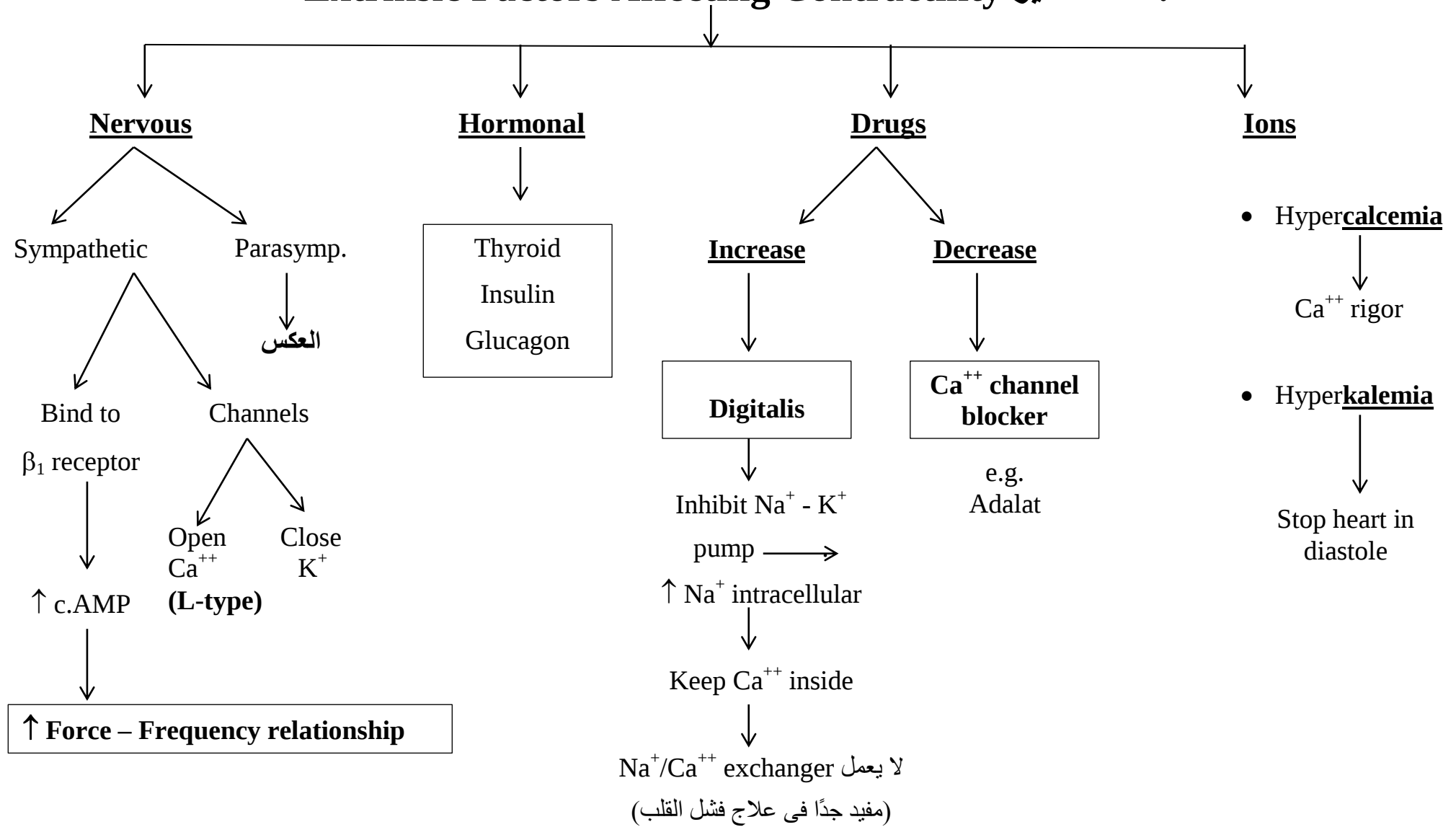
Conductivity



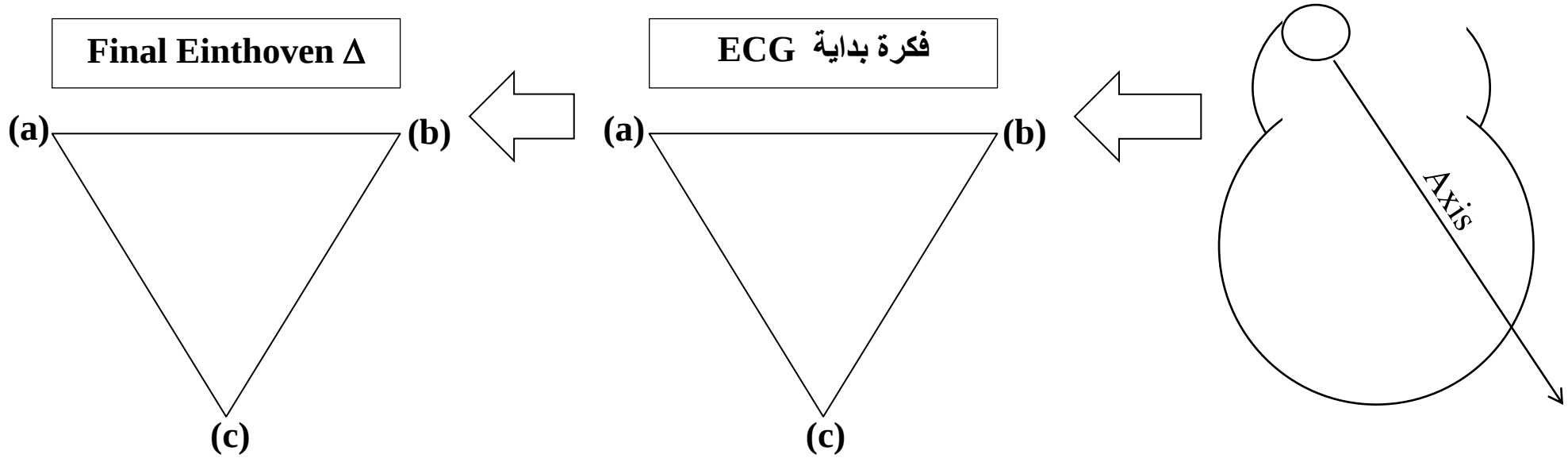
د. محمد فايز Factors Affecting Contractility



د. محمد فايز Extrinsic Factors Affecting Contractility



د. محمد فايز Einthoven Triangle



أساس فكرة رسم القلب:

Axis of heart is directed downward, to left

∴ Einthoven Δ

معظم الوصلات ناحية الشمال

يمكن تسجيل كهرباء الناتجة من القلب

لأنه يقع على ابعاد متساوية من ٣ نقاط:

- a) Rt shoulder.
- b) Lt shoulder.
- c) Symphysis pubis.

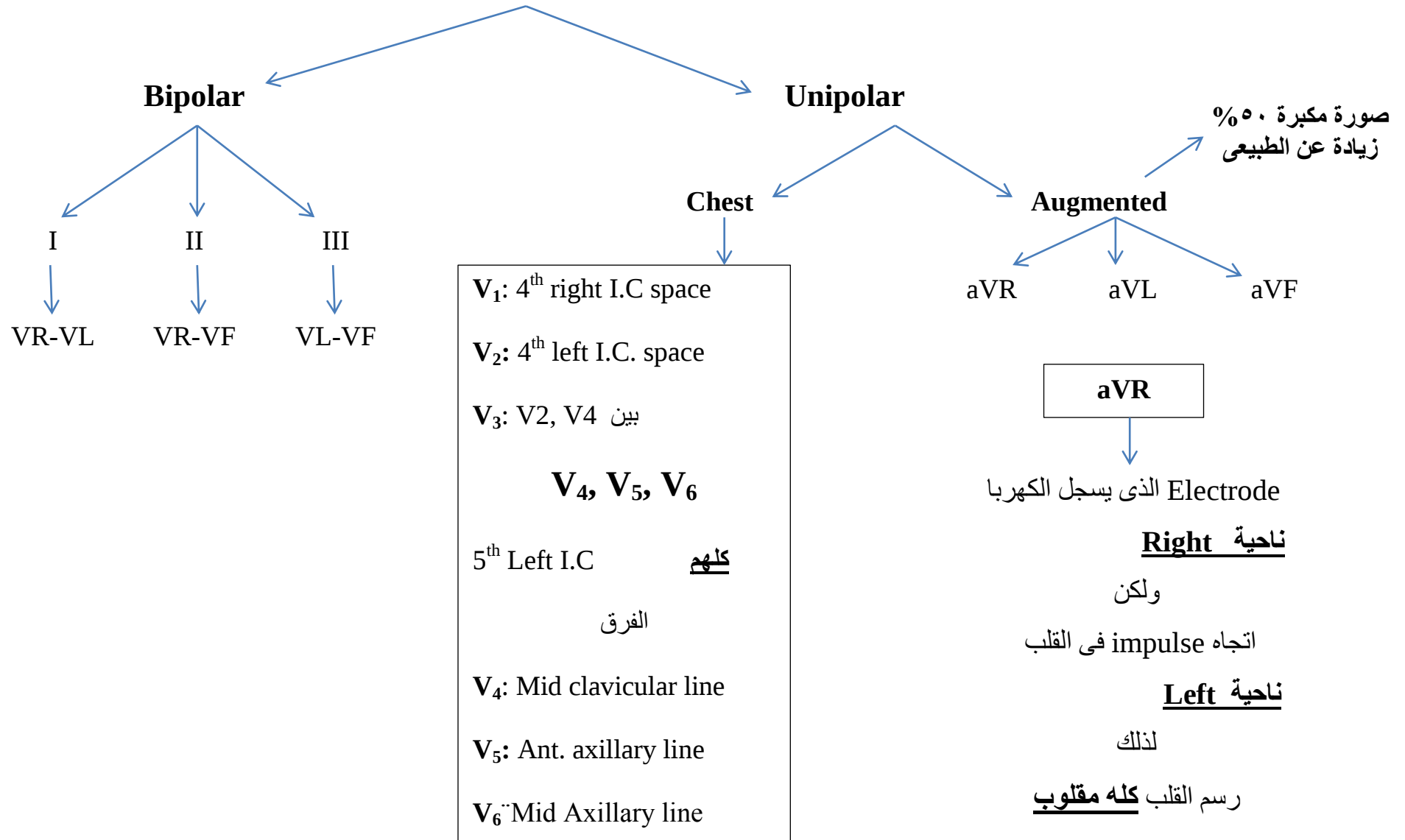
تم تغيير ٣ نقاط

∴ Final Einthoven Δ:

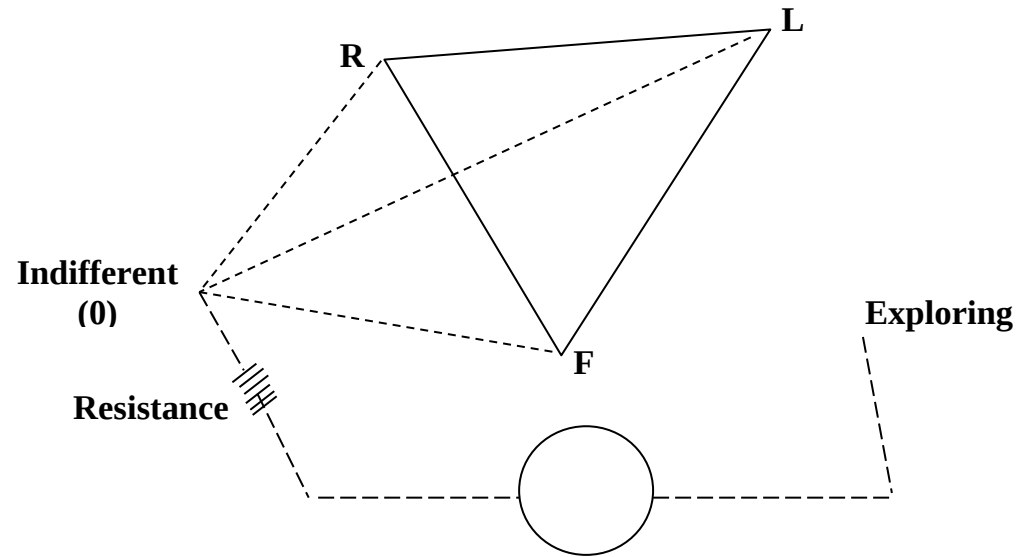
- a) Rt arm.
- b) Lt arm
- c) Lt foot

معظم الوصلات
شمال

د. محمد فايز ECG Leads



Unipolar Lead



Unipolar lead معناها أسجل الكهربيا عند نقطة واحدة (**Exploring**) أما النقطة الأخرى (**In different**) تساوى صفر

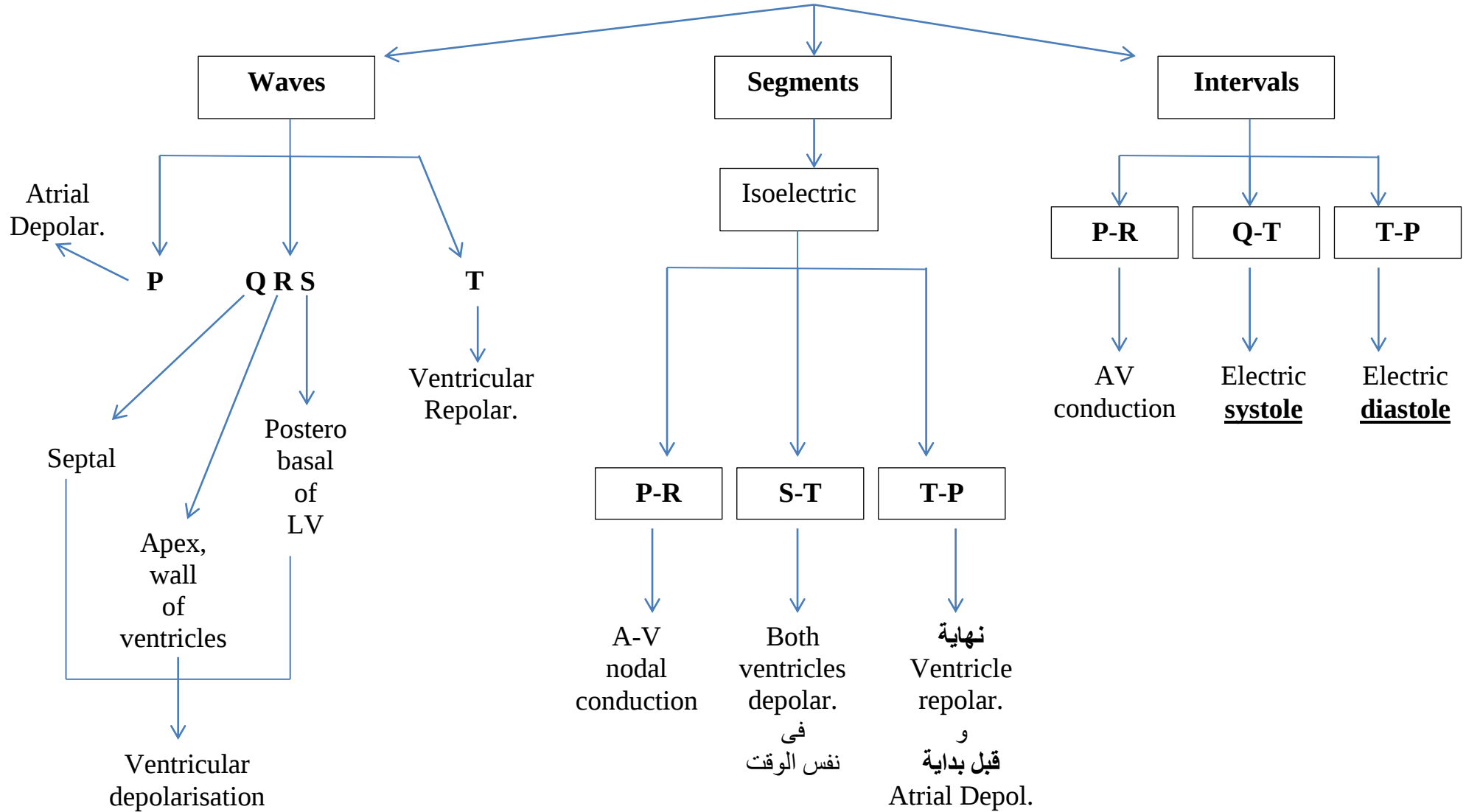
$$\text{Indifferent} = (R - L) + (L - F) + (F - R) = 0$$

لا توجد نقطة في جسمك، الكهربيا عندها تساوى صفر

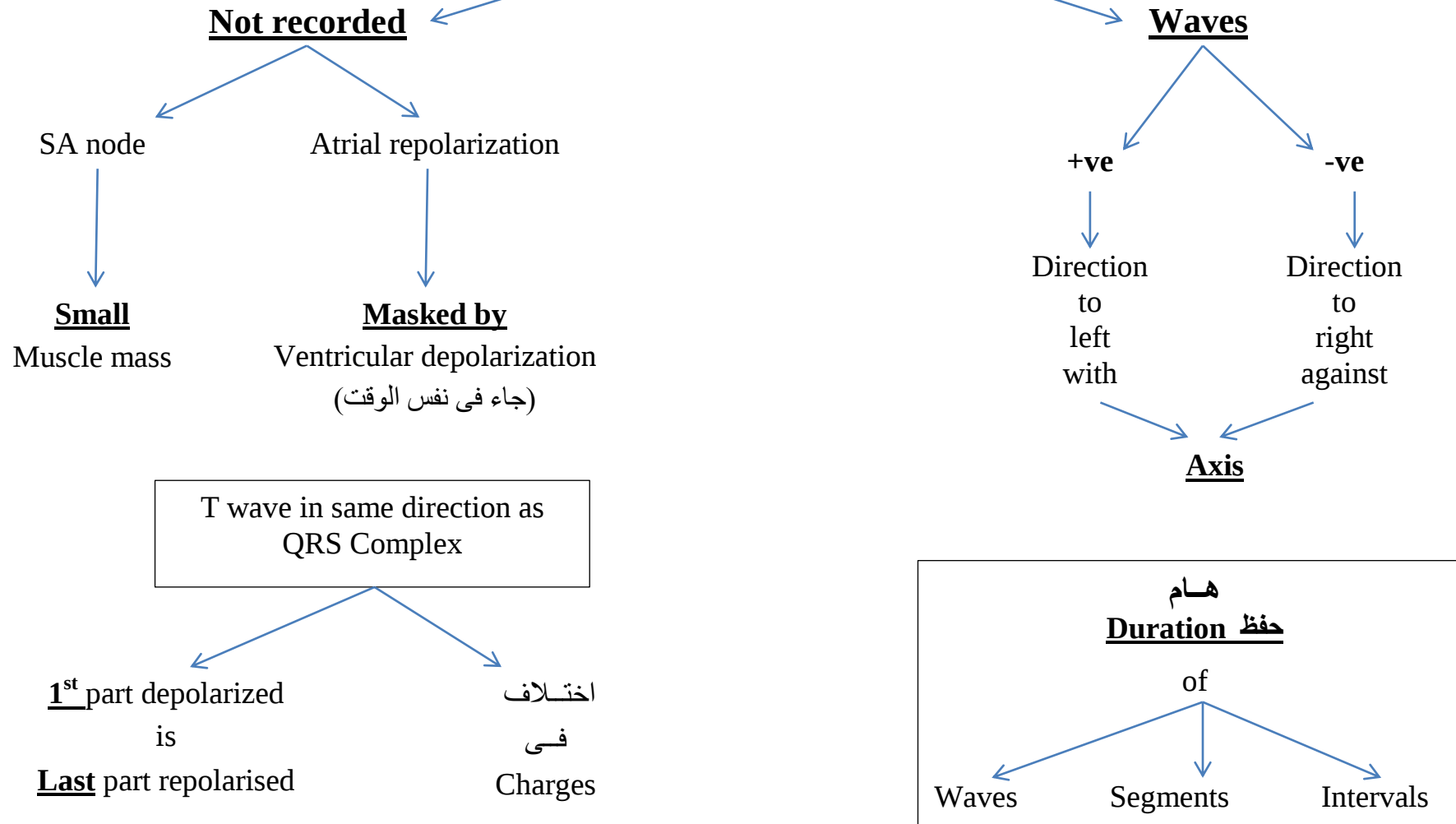
It can be applied only by **high resistance (5000 ohm)** to be indifferent

Exploring electrode
 ↗ Limb (**unipolar Limb Lead**)
 ↘ Chest (**unipolar chest Lead**)

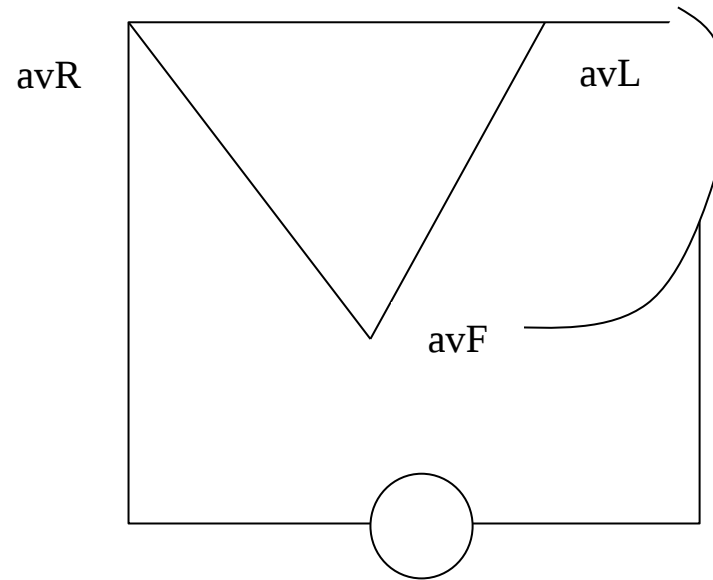
د. محمد فايز Normal ECG



ECG Notes د. محمد فايز



Proof: Augmented Unipolar Leads = 50% ↑ Unipolar Lead د. محمد فايز



$$avR = VR - \left(\frac{VL + VF}{2} \right)$$

$$\therefore 2 avR = 2 VR - (VL + VF)$$

$$\Delta VR + VL + VF = \text{Zero (Kirchhoff's 2nd law)}$$

$$\therefore (VL + VF) = -VR$$

$$\therefore 2 avR = 2 VR - (-VR) = 3 VR$$

$$\therefore \underline{avR = 3/2 VR}$$

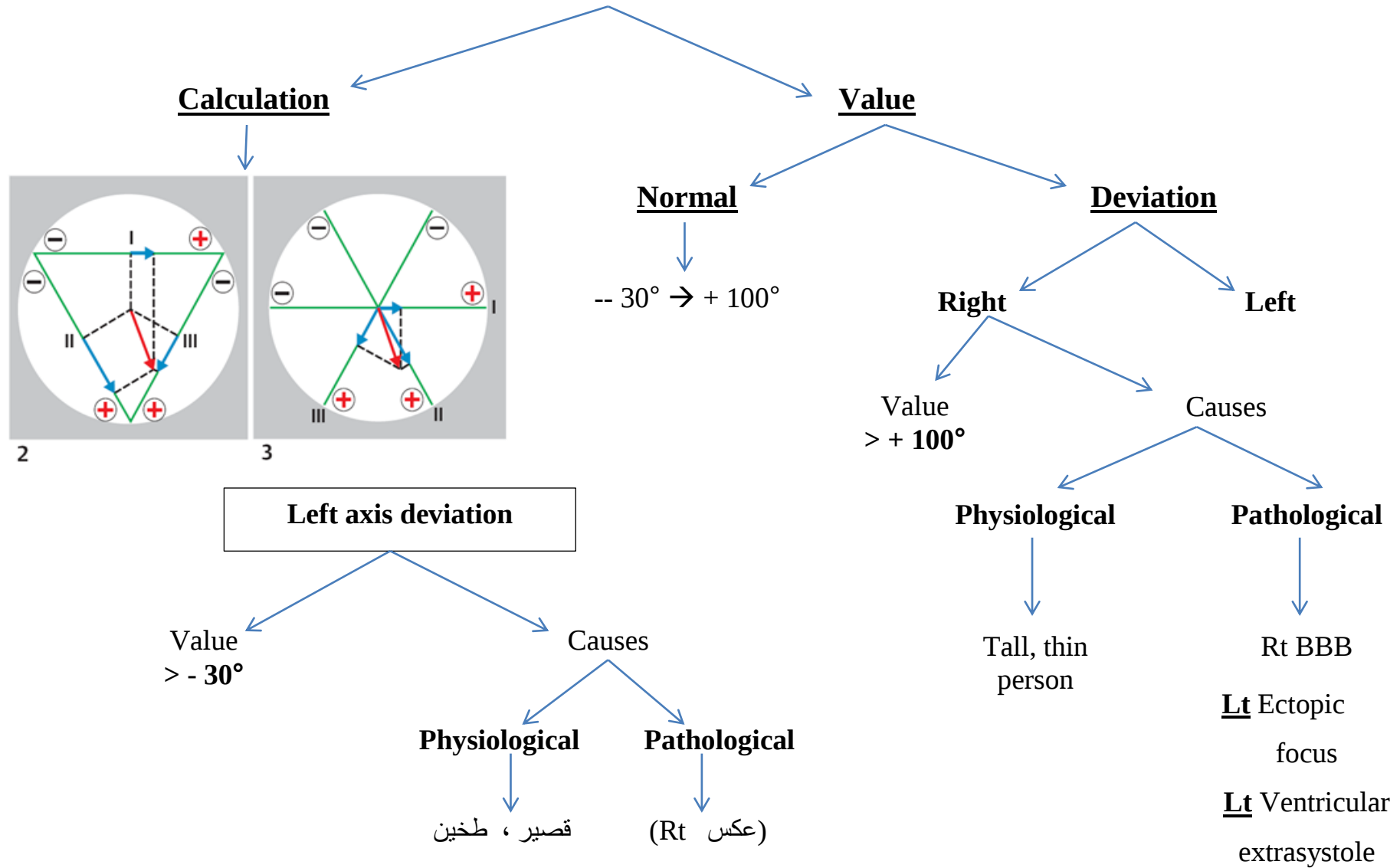
د. محمد فايز Notes on ECG

	Atrium		Ventricle	
	Endocardium	Epicardium	Endocardium	Epicardium
Depolarised	1 st	Last	1 st	Last
Repolarised	1 st	Last	Last	1 st

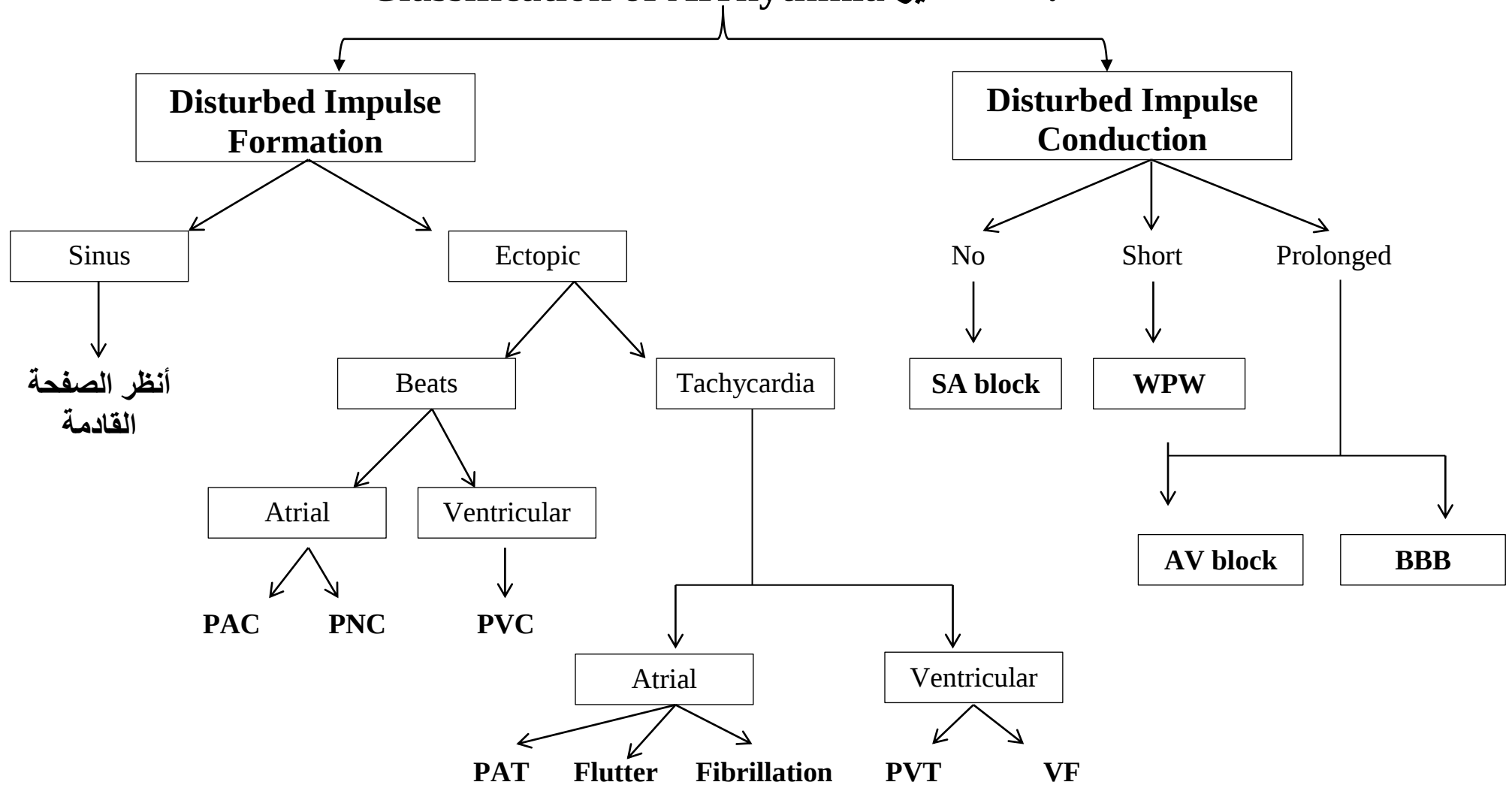
Premature atrial contractions **not** followed by compensatory pause

Premature ventricular contractions **followed by compensatory pause**

د. محمد فايز Cardiac Axis



Classification of Arrhythmia د. محمد فايز



د. محمد فايز Sinus Arrhythmia

	Sinus Tachycardia	Sinus Bradycardia	Respiratory sinus arrhythmia
Heart rate	>100 bpm	<60 bpm	↑ (Inspiration) ↓ (Expiration)
Causes	1. Sympathetic 2. Regulation of ↓ BP 3. Hyperthyroidism 4. Fever	1. Vagal tone 2. Regulation of ↓ BP 3. Hypothyroidism 4. β blockers	<u>Inspiration</u> Stretch receptors (Lung) ↓ ↓↓ CIC ↓ ↓↓ Vagal tone ↓ ↓ Heart rate
ECG	N o r m a l ← Short c. cycle ↓ Long c. cycle → Short c. cycle (Inspiration) والعكس (Expiration)		

د. محمد فايز Ectopic Focus

W

What ? **Strong** Stimulus

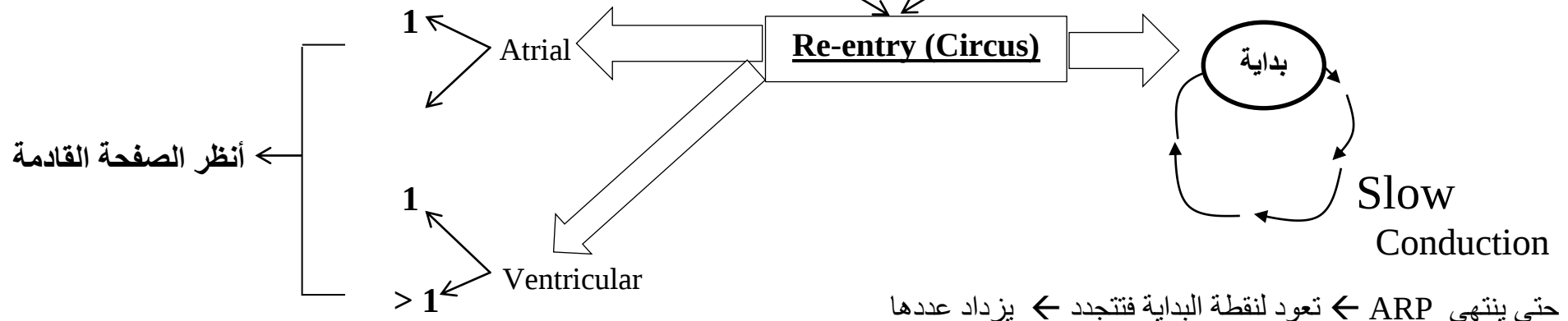
Where ? **AV node/Septum/wall**

Why ? Inhibits SAN (**over drive suppression**)

When ?

RRP يبدأ

ARP ينتهي



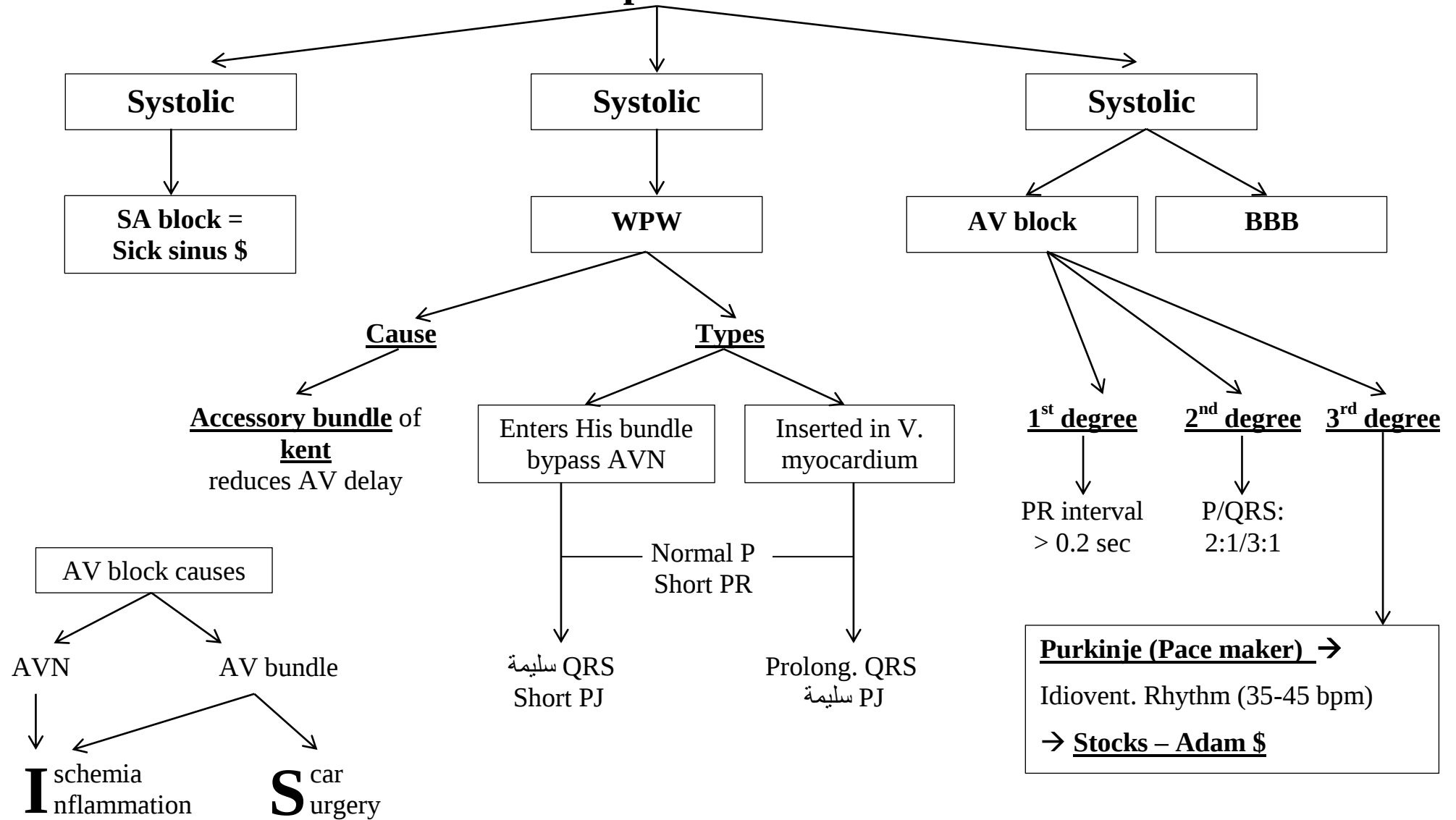
د. محمد فايز Ectopic beats

	PAC	PNC	PVC (V. extrasystole)
Origin		AV node	1) His-Purkinje 2) Ventricular wall
E C G			
P wave	A b n o r m a l		تختفى Before After
PR interval	Short	Short or absent	
QRS complex	Normal	Normal	1. High voltage 2. Wide prolonged (Bizarre)
Compensatory pause	<u>Un</u> common بعدها		Common بعدها
T wave	Normal	Normal	Inverted
Axis	Normal	Normal	ينحرف للناحية العكسية
Pulse	Weak		<u>Premature</u> : weak <u>Post extrasystole</u> : strong

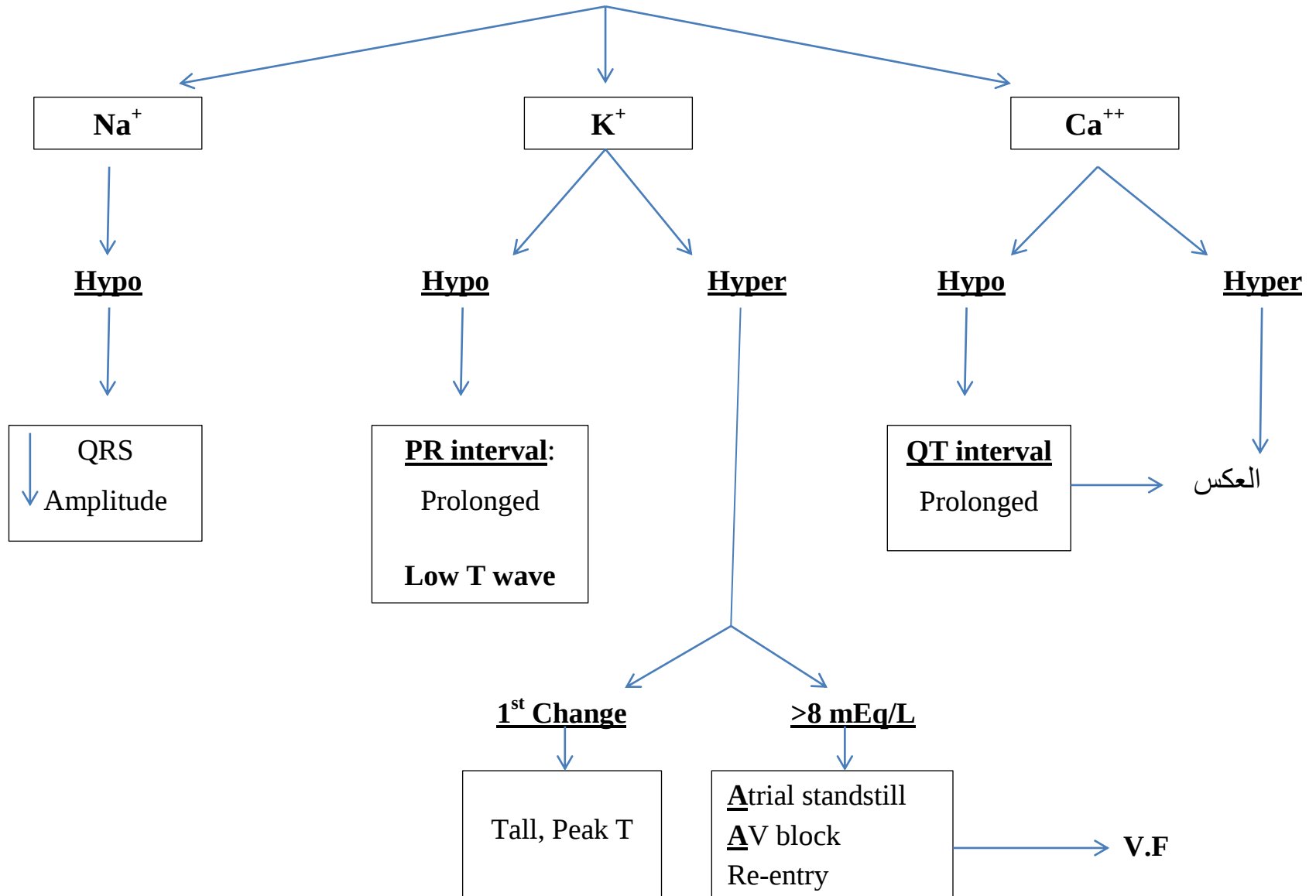
د. محمد فايز Ectopic Tachycardia

	PAT	PNT	PVT	A. flutter	A.F	V.F
Cause						1. Ectopic 2. صدمة كهربية 3. Ischemia
Character-ised by	<u>Heart rate:</u> 140-220 دائما تحدث فى الشاب السليم	<u>Heart rate:</u> 150-220 صعب تفرقها عن PAT	<u>Heart rate:</u> 150-250 لاي وجد أى دقات طبيعية فيما بينها	<u>A. Rate:</u> 250-350 <u>V. rate:</u> 125-175 <u>Rhythm:</u> Regular Irregular	<u>A. rate:</u> 350-500 <u>V. rate:</u> 100-150 <u>Rhythm:</u> I rregular rregular	<u>Vulnerable period:</u> Coincide which mid part of T wave
E C G						
P	Abnormal		تختلفى	2:1 / 3:1	(F wave)	Bizzar
QRS	Normal		Slurred, Notch, Wide	Normal		
T	Normal		Opposite to QRS	Normal		
Contraction				Synchronus	A synchronous (Bag of worm)	
Seriousness	Not serious		Serious → VF	Not serious		Serious (مميتة)
Treatment	↑ <u>Vagal Tone</u> ↙ ↘ Carotid Massage Oculo-cardiac reflex		<u>Cardioversion</u>	As <u>PAT</u> , <u>PNT</u>		As <u>PVT</u>

د. محمد فايز Disturbance in Impulse Conduction



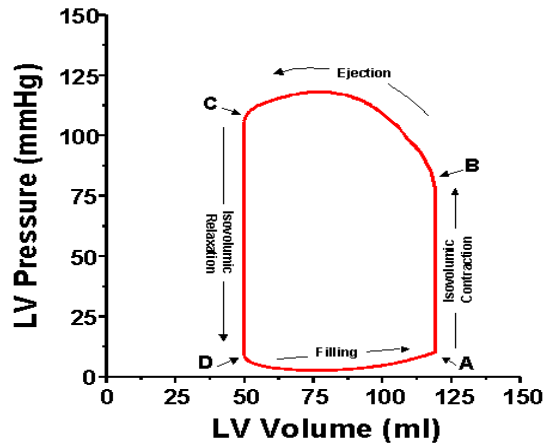
د. محمد فايز Effect of Ions on ECG



د. محمد فايز Cardiac Cycle

	Atrial systole	Isovol. contraction	Maximum ejection	Reduced ejection	Proto diastolic	Iso vol. relaxation	Rapid filling	Slow filling
Atrial Pr.	↑	↑	↓	Increased (VR)			↓	No change
Ventricle Vol.	↑	Constant	↓	↓	↓	Constant	↑	↑ <u>Slow</u>
Ventricle Press.	↓	↑	↓	↓	↓	↓	↓	↑ <u>Slight</u>
Aortic Press.	↓	↓	↑	↓	↑	↑ بداية ثم يقل	↓	↓
CBF	↓	↓	↓	↓	↓	↑	↓	↓
Valves	<u>Open</u> (A-V) <u>Close</u> <u>Semi</u> <u>lunar</u>	<u>All closed</u>	<u>Open</u> : Semilunar <u>Close</u> : A-V valves			<u>All closed</u>	<u>Open</u> : A-V <u>Closed</u> : Semilunar	
Heart sounds	4 th	1 st	1 st	-	-	2 nd	3 rd	-
ECG	P-wave before (0.02 sec)	* Q-wave before (0.02 sec) * QRS	ST segment T بداية	1 st ½ T-wave		End T-wave TP segment بداية	T-P segment	

د. محمد فايز LV Pressure Volume Loop



Significance

1. Area under curve = SW

$$= \underline{\underline{SV \times MAP}}$$

2. BC represents SV

3. DA represents ventr. Filling.

4. RV pressure volume loop (as LV)

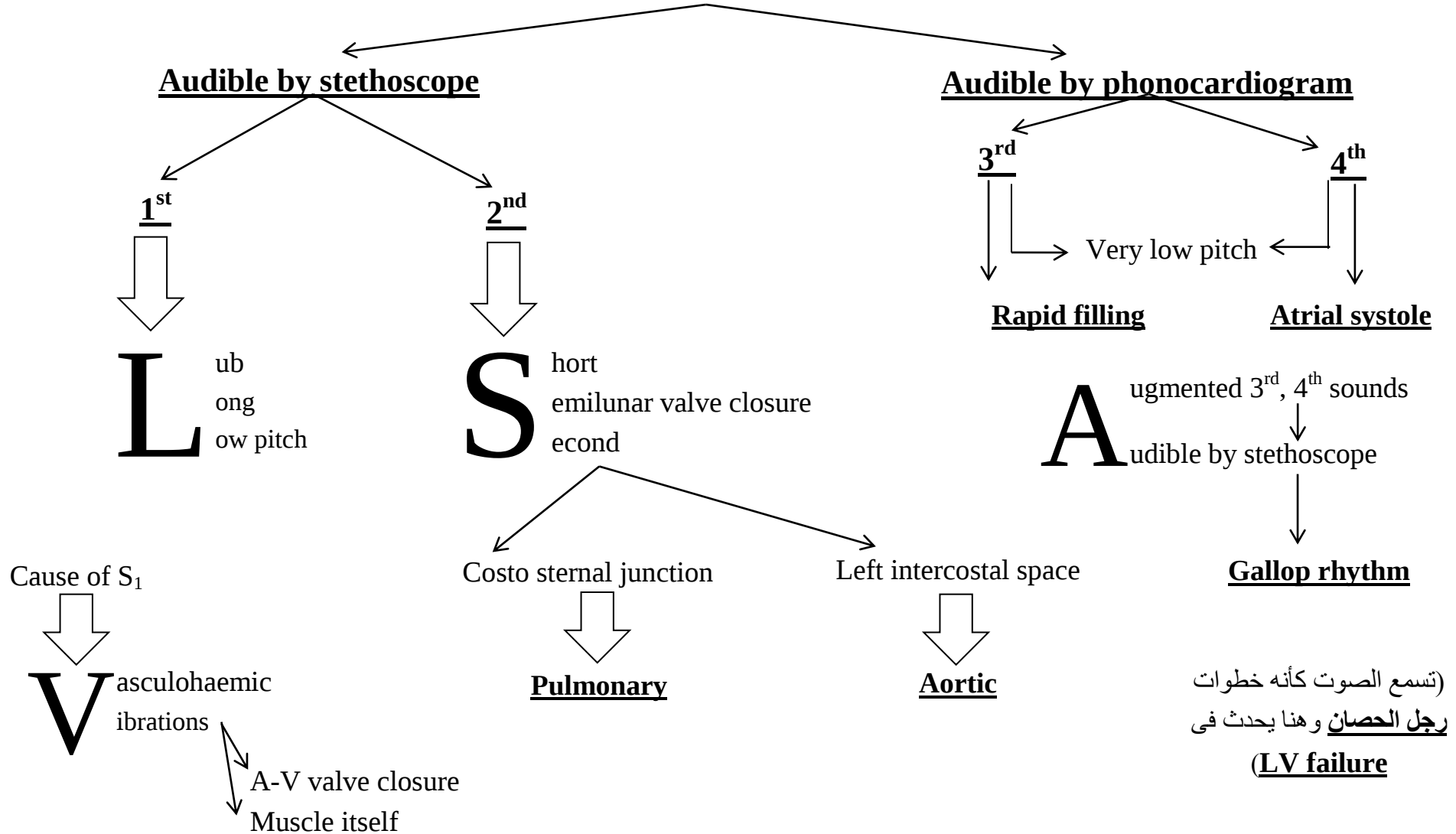
(Systolic RV pressure: 25 mmHg)

5. Heart failure:

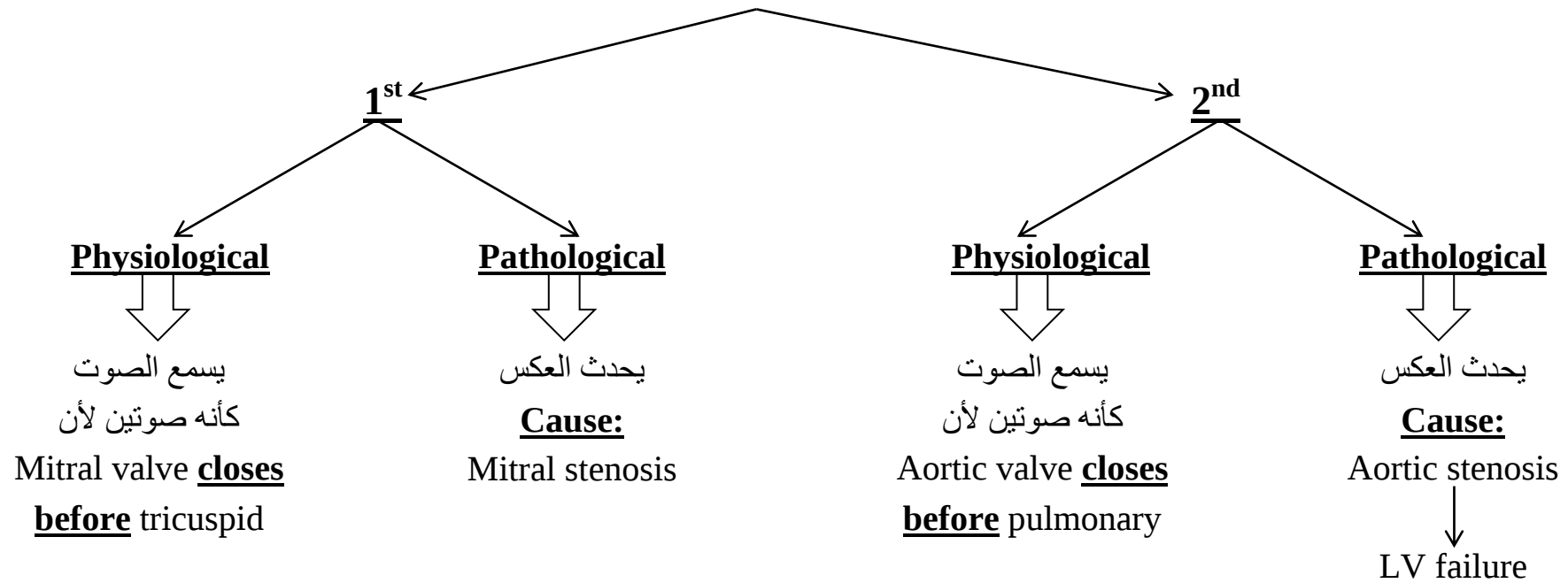
Contractility curve (Shirt to Rt)

Points				
	A	B	C	D
Valves	MVC	AVO	AVC	MVO
Lines				
	AB	BC	CD	DA
Phase of c. cycle	Isovolumetric contraction	<u>Rapid</u> , <u>Reduced</u> ejection	Isovolumetric relaxation	Rapid, slow filling
Ventricular volume	Constant	↓	Constant	↑
Ventricular pressure	↑	<u>Rapid ejection</u> ↑ (80-120 mmHg) <u>Reduced ejection</u> يقل	↓ ↓ ↓	<u>Rapid filling</u> يقل حتى يصل صفر <u>Slow filling</u> ↑ up to (5-8 mmHg)

Heart Sounds د. محمد فايز



د. محمد فايز Splitting of heart sounds



Cause:

Inspiration → ↑ VR to RV → Delay closure of tricuspid valve

Physiological splitting 'S₂'

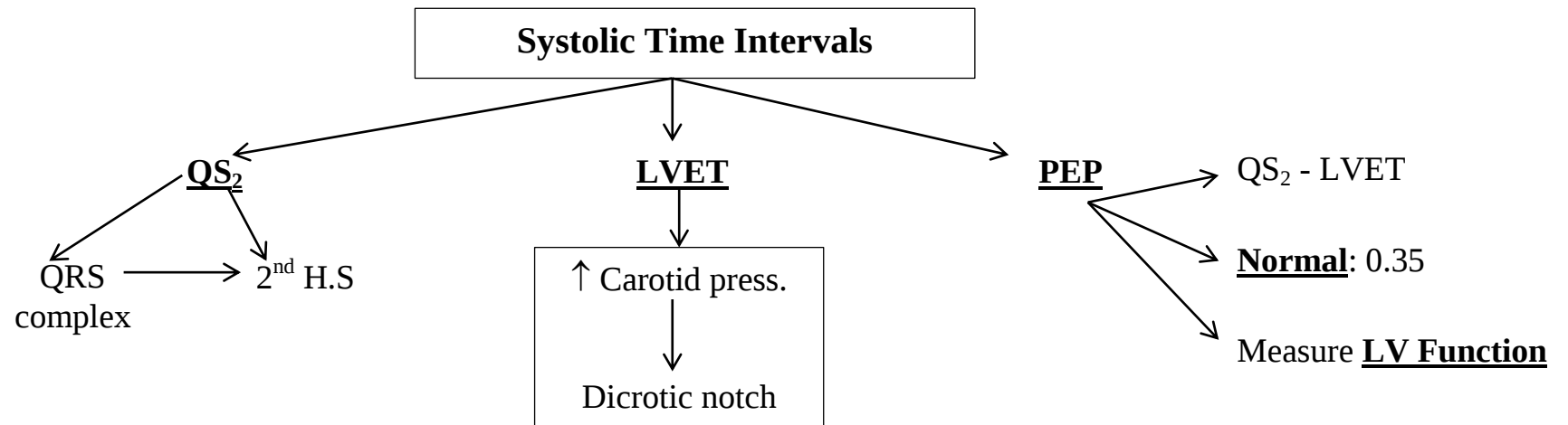
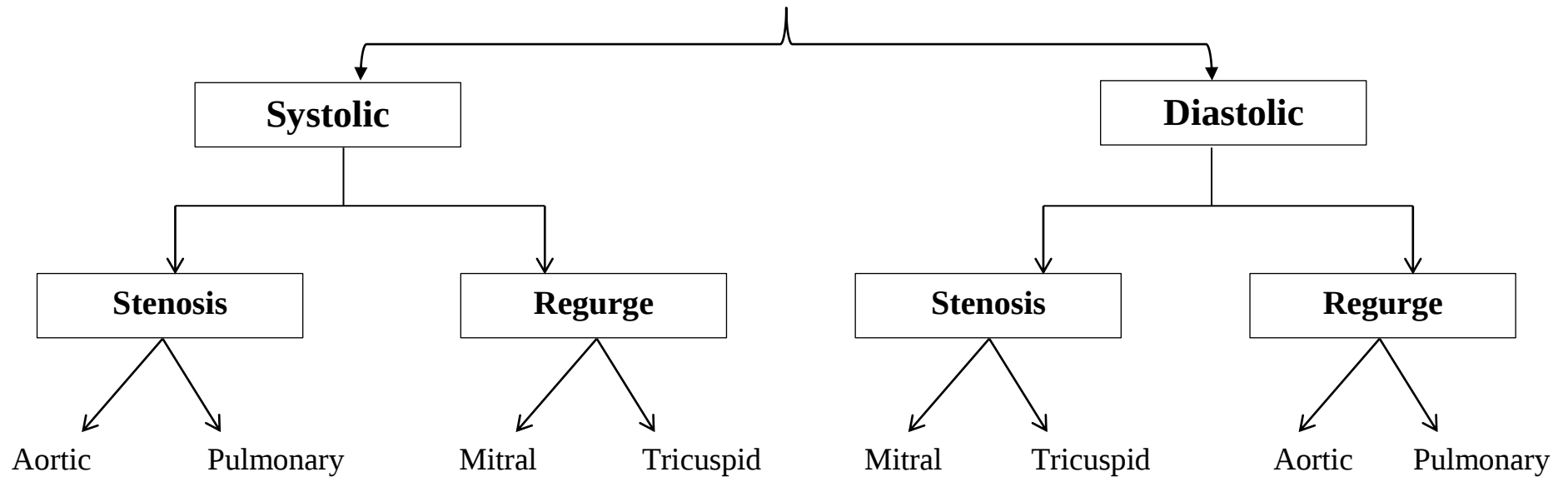
→ Easy heard

→ **Than splitting of S₁**

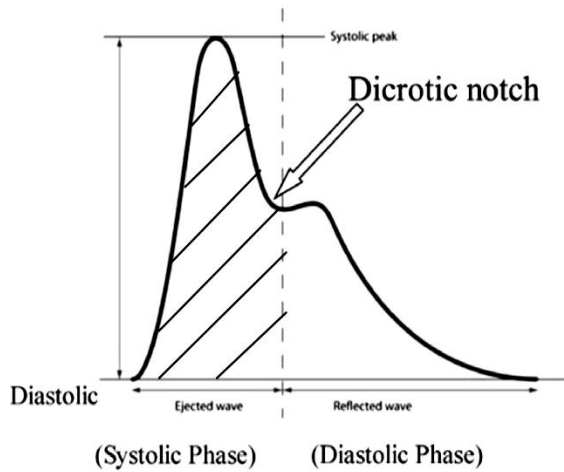
→ **Pulmonary area**

→ **Children**

د. محمد فايز Murmurs

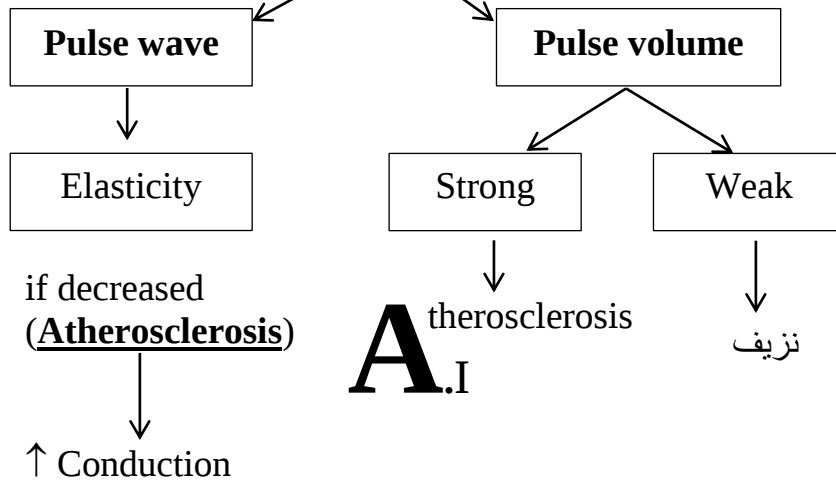


د. محمد فايز Arterial Pulse Wave

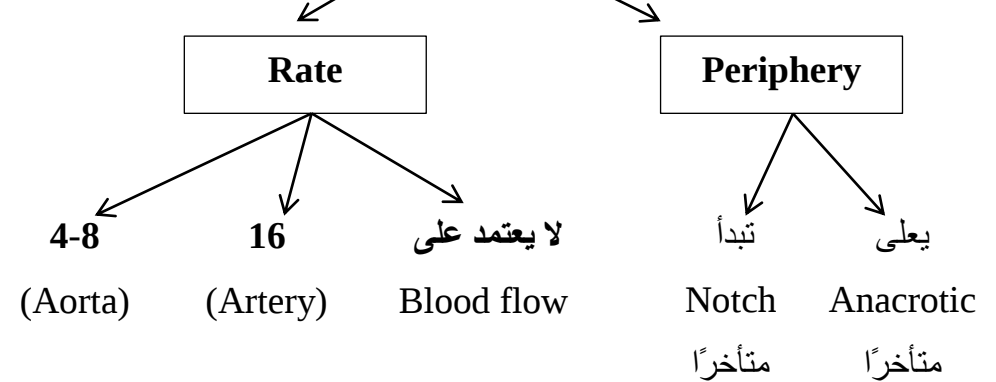


	Anacrotic limb	Catacrotic limb	Dicrotic notch (Incisura)	Dicrotic wave
Aortic pr.	↑	↓	↑	↑
Cause	Opening of Aortic valve	Less vibrations Set up in Aorta	Sudden AVC	Aortic Elasticity
Phase of c. cycle	Maximum ejection	* Reduced ejection * Ventricular diastole	نهاية Protodiastolic	Iso vol. relaxation

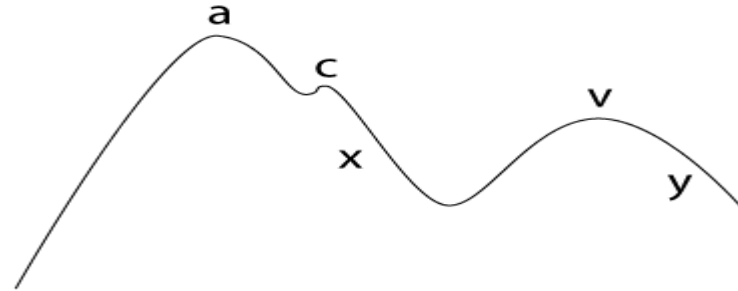
Factors affecting



Notes



د. محمد فايز (JVP) Jugular Venous Pulse



	"a" wave	"c" wave	"x" descent	"v" wave	"y" descent
Wave	+	+	-	+	-
Cause	Atrial ms contract ↓ ↑ Atrial press. ↑ JVP	Bulge of <u>Tricuspid valve</u> Into Rt atrium	Down ward Displacement of AV ring	VR While <u>Tricuspid valve</u> is closed	Opening of <u>Tricuspid valve</u> and Rapid emptying
Phase	Atrial systole	Iso volumetric contraction	Rapid ejection	Isovolumetric relaxation	Rapid filling
Signif- icance	No → AF Large → Tricuspid stenosis Giant → 3rd AV block	Giant → Tricuspid regurge	Note a-c interval prolonged ↓ 1st degree AV block		

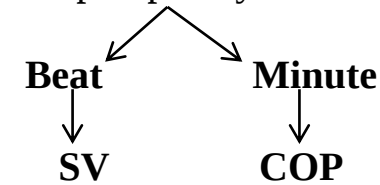
د. محمد فايز COP

$$\text{COP} = \text{SV} \times \text{HR}$$

$$\text{COP} = (\text{EDV} - \text{ESV}) \times \text{HR}$$

هامة Definitions

* Volume of blood pumped by each ventricle

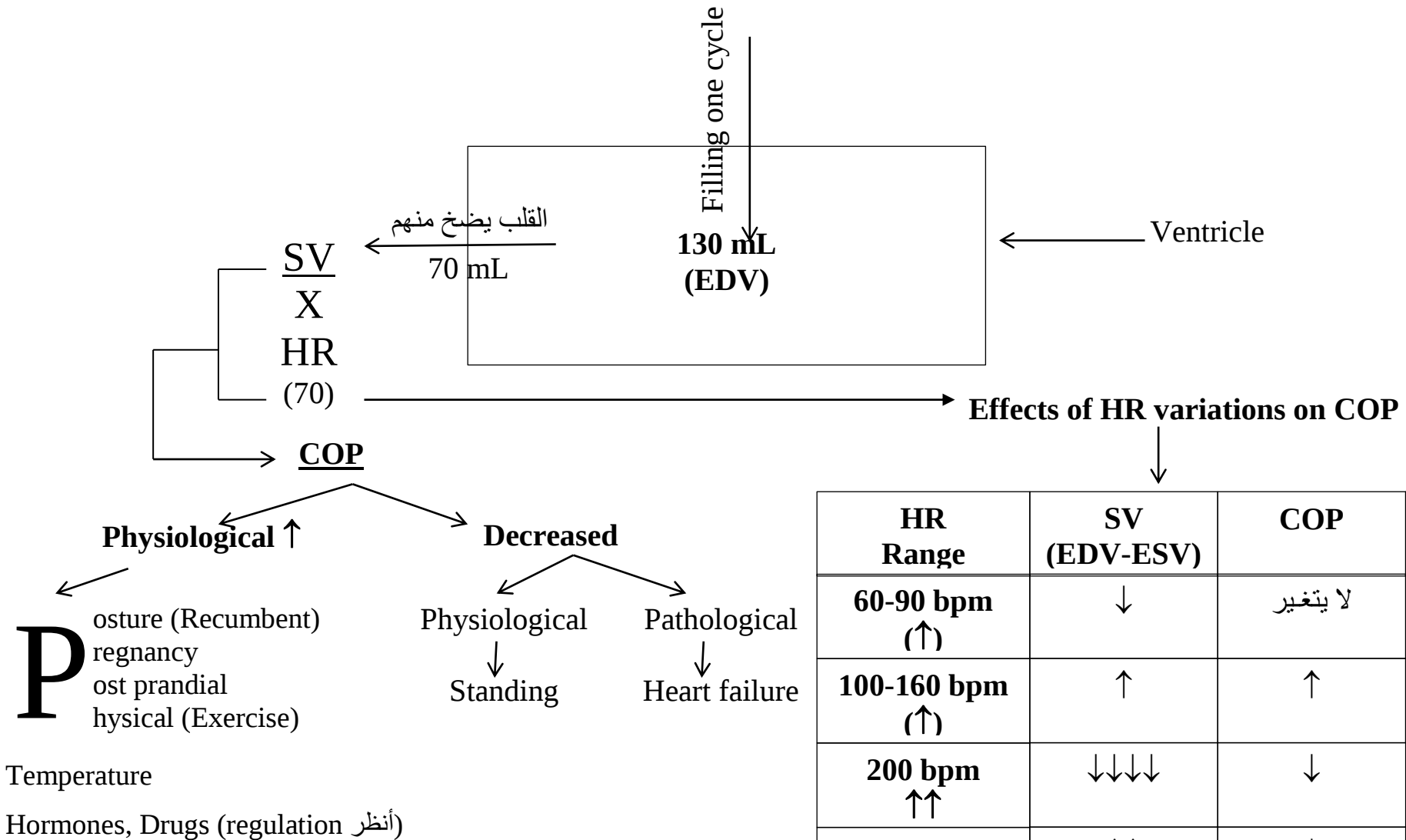


* **Cardiac Index** = $\text{COP} / \text{SA} = 3.2 \text{ L/m}^2$

* **Ejection Fraction (EF)** = $\text{SV} / \text{EDV} \times 100 \text{ (N=55\%)}$

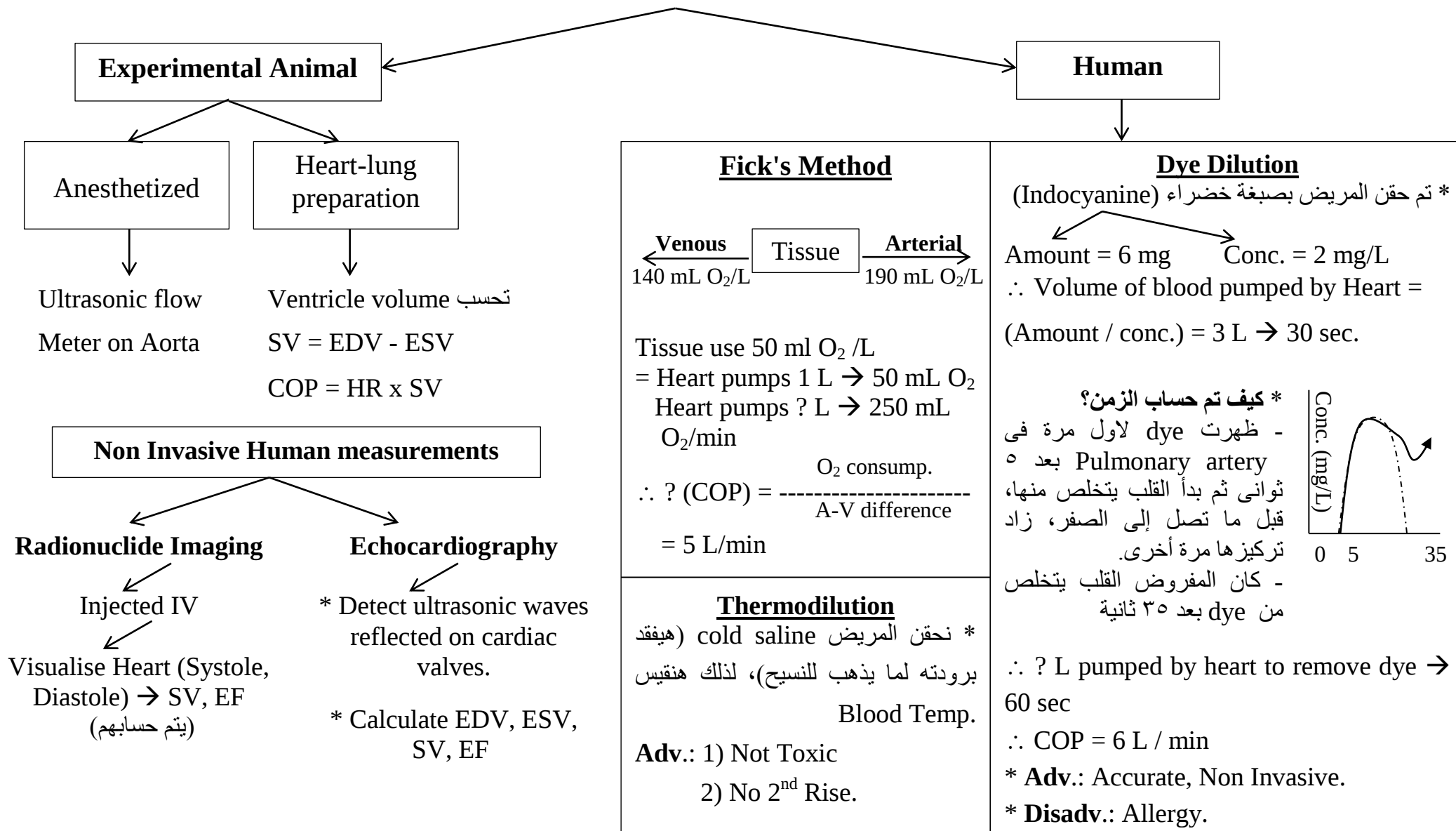
	EDV	ESV	HR
Rest	130 mL	60 mL	60-90 bpm
Exercise	240 mL	30 mL	180 bpm
Limited by	Starling Law	N° of catecholamine vesicles	Filling Time
↑	Physiological ($\uparrow$ Venous Return = VR) 1. $\uparrow$ Blood volume 2. Venous tone. 3. Skeletal muscle contraction 4. Intrathoracic negativity 5. Atrial contraction 6. Ventricular compliance	Physiological $\downarrow$ Vagal Tone (-ve Inotropic)	أنظر HR Regulation (Vascular)
↓	Pathological Pericardial effusion $\downarrow$ Cardiac Tamponade Heart failure MI	Pathological $\downarrow$ Heart Failure	
		Physiological $\downarrow$ Exercise	

د. محمد فايز Cardiac Output (COP)



HR Range	SV (EDV-ESV)	COP	Notes
60-90 bpm (↑)	↓	لا يتغير	واحد زاد واحد قل
100-160 bpm (↑)	↑	↑	الاثنين زادوا
200 bpm ↑↑	↓↓↓↓	↓	في الحالتين: النقص أكبر من الزيادة لذلك يقل COP
40 bpm ↓↓↓↓	↑↑	↓	

د. محمد فايز COP Measurements



Regulation of COP د. محمد فايز



	Heterometric	Homeometric
Phenomenon	Pre Load	After Load
Stimulus	↑ VR	
Time	يبدأ أولاً Transient (2-5 minutes)	ثانياً Prolonged (Not Time Limit)
EDV	↑	Constant
ESV	↓	↓
SV	Increased	
Significance	Physiological (Exercise) Pathological (Heart Failure)	

Heterometric = Pre Load = $\uparrow$ EDV = Starling law

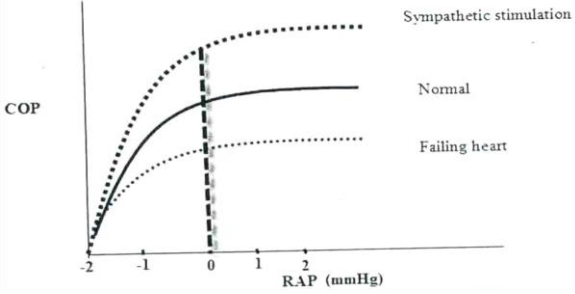
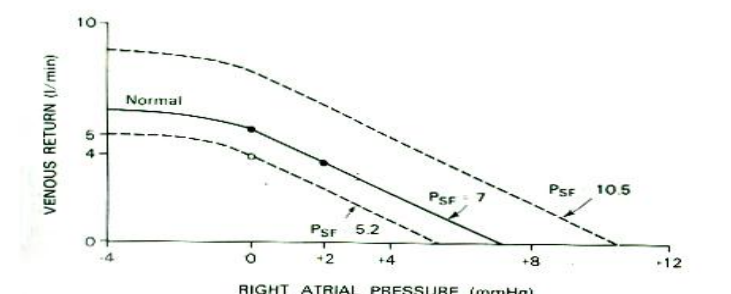
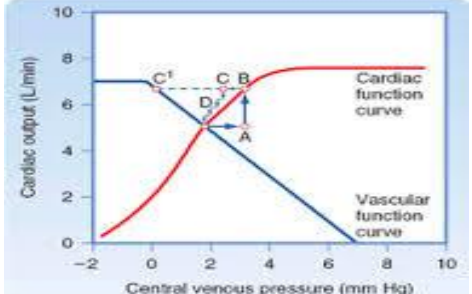
	↑	↓
1. Nervous	Sympathetic Ino chrono ↙ ↘ ↘ ↙ Tropic ↓ β receptor $\rightarrow$ $\uparrow$ c.AMP	Parasymp. العكس
2. Hormonal	Glucagon $\uparrow$ c.AMP ↘ ↙ ↘ Tropic Ino chrono (strong) (weak)	
3. Drugs	$\uparrow$ c.AMP ↙ ↘ β adrenergic agonist inhibit break down ↓ Caffeine Theophylline $\uparrow$ Ca^{++} intracellular: Digitalis	$\downarrow$ c.AMP ↓ β Blockers ↓ Ca^{++} ↓ Ca^{++} channel blockers (Adalat)
4. Ions		Hypoxia ypercapnia yperkalemia

د. محمد فايز Compare Between MCP, CVP

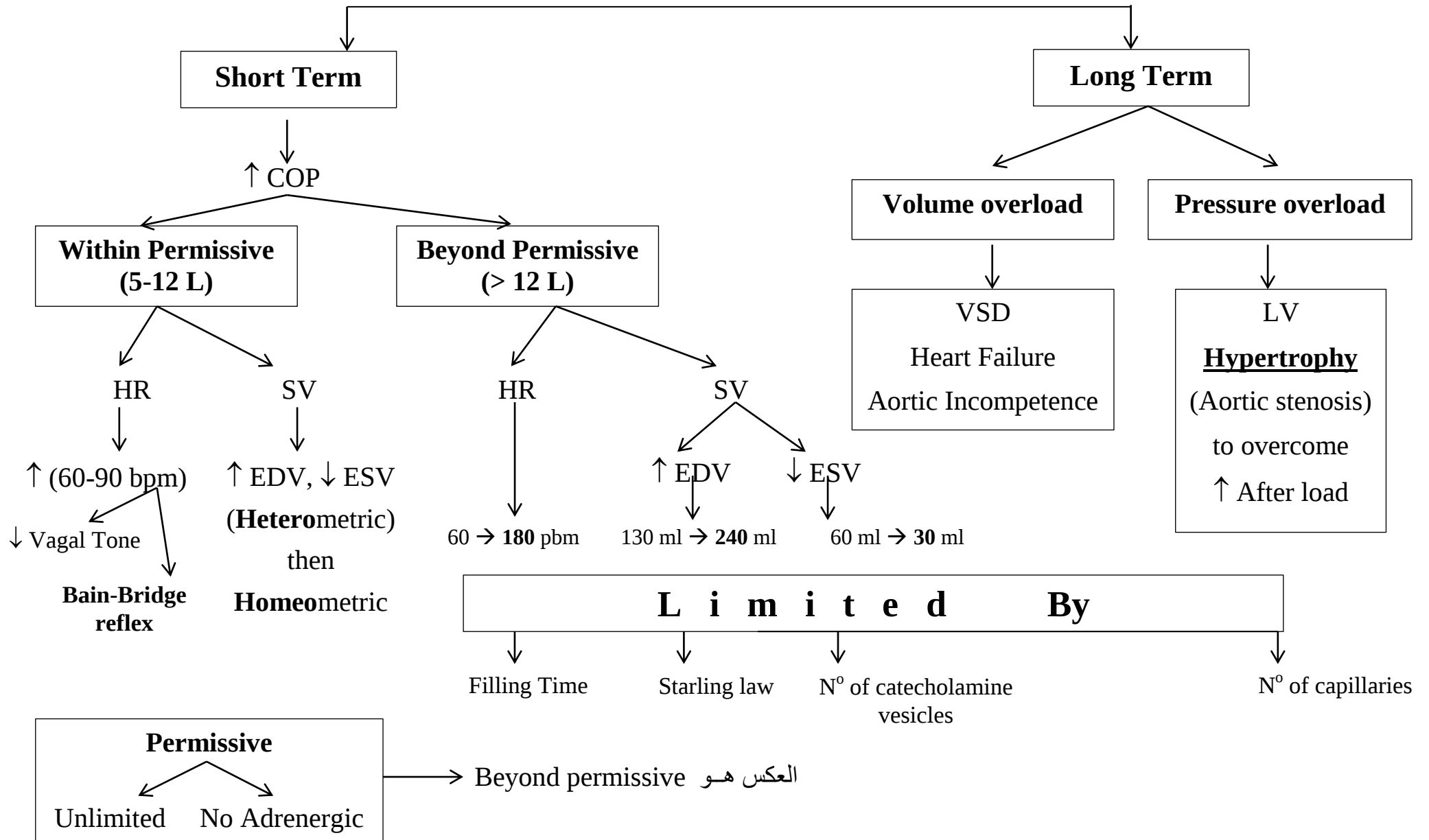


	Mean Circulatory Pressure (MCP)	Central Venous Pressure (CVP)	
Definition	<u>Average</u> pressure (Intravascular)	<u>Pressure in thoracic veins</u> (متصلة بالقلب)	
Normal	7 mmHg	0-5 mmHg	
	Static	Dynamic	
Determinants	$V_{\text{venous capacity}}$	V_{R} entricular Pump	
		Physiological	Pathological
Decreased	↓ Blood volume ↑ Venous capacity	Gravity Inspiration	Venodilatation Shock
		Physiological	Pathological
Increased	↑ Blood volume ↓ Venous capacity (Venoconstriction)	<u>E</u> xpiration (Forced) <u>S</u> ympathetic	<u>H</u> ypervolemia eart Failure <u>E</u> xcess blood Transf. mbolism (Pulmonary) <u>S</u> hock (Cardiogenic)

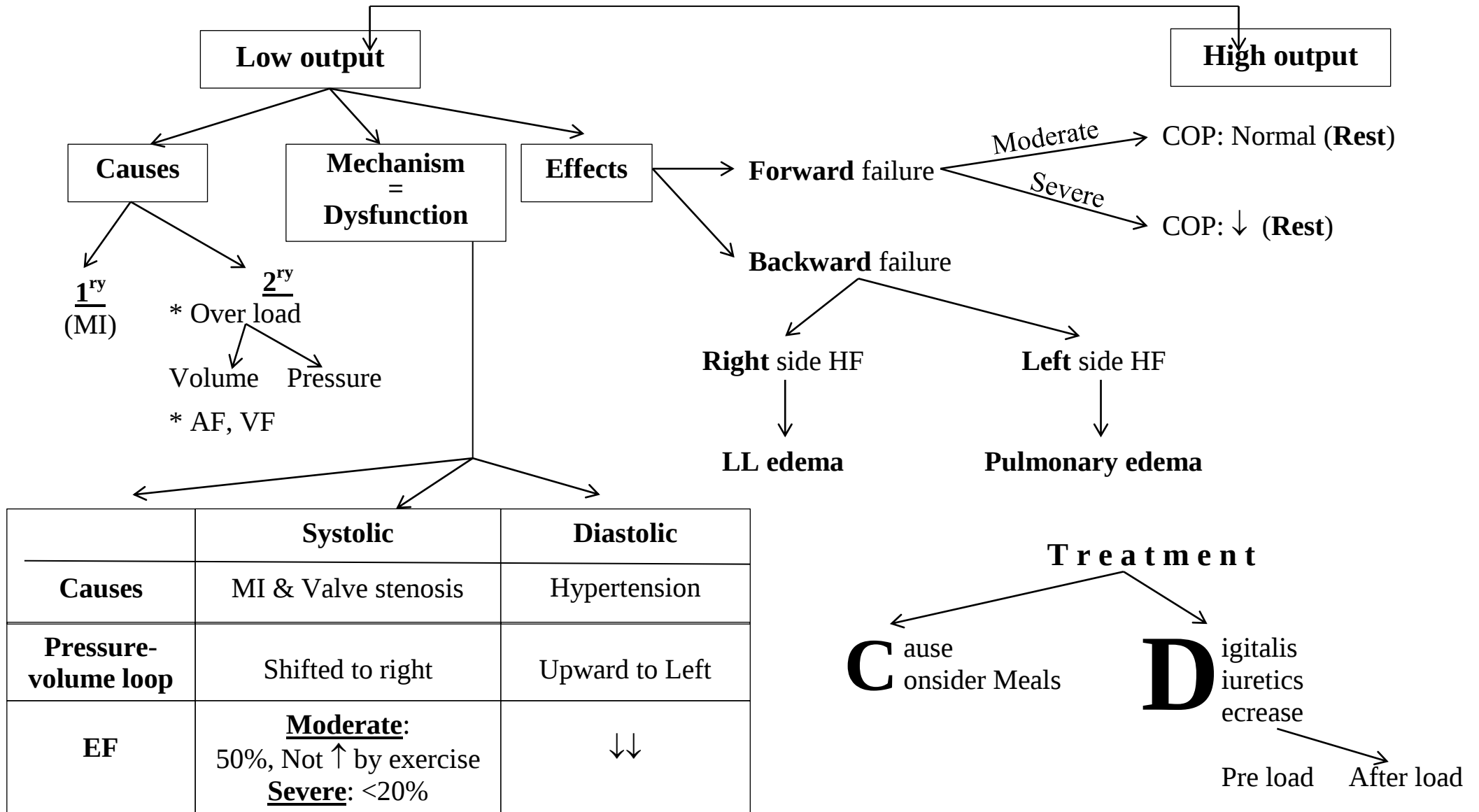
د. محمد فايز Function Curves

	Ventricular	VR (Vascular)	Combined (Ventricular, VR)
			
Relation	Direct (RAP, COP) RAP (Preload on Horizontal) COP (Vertical scale) كلما يزداد RAP ← يزداد COP (Unlimited)	Inverse (VR, RAP) RAP (Horizontal scale), VR (Vertical) VR = MCP - CVP Static 7 mmHg (عندما يتوقف القلب، يصبح الضغط في كل circulation متساوي) CVP: Pressure in great veins (مدخل القلب) = RAP = 0-5 mmHg * يزداد VR كلما يقل RAP حتى يصبح RAP يساوي Gradual collapse of veins: السبب: (-2 mmHg) * يتوقف VR عندما يتساوى CVP, MCP	VR or COP (Vertical scale) RAP (Horizontal scale) Equilibrium point: Point of intersection 2 curves.
Shift to right	Downward (-ve Inotropic) ↘ Vagal ↘ Heart failure	Upward (↑ MCP due to veno constriction)	-ve Inotropic (Heart failure)
Shift to left	Upward (+ve Inotropic = Sympathetic)	Downward (↓ MCP due to venodilatation, Hge).	+ve Inotropic (Sympathetic)

Cardiac Reserve د. محمد فايز



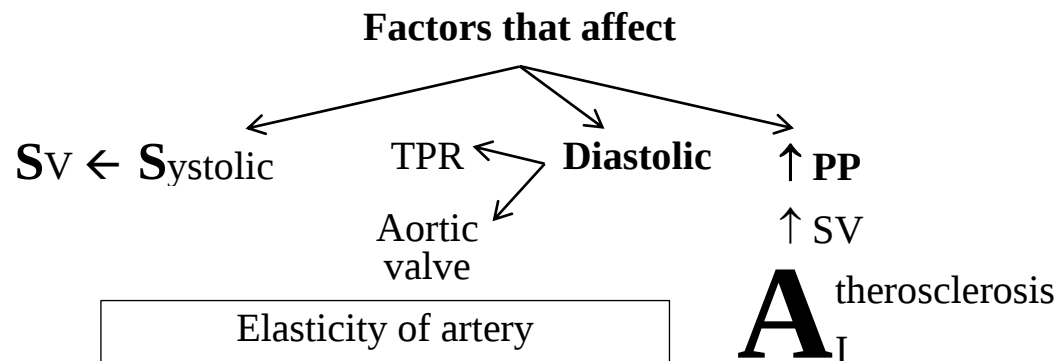
د. محمد فايز (HF) Heart Failure (HF)



Vascular

د. محمد فايز (ABP) Arterial Blood Pressure

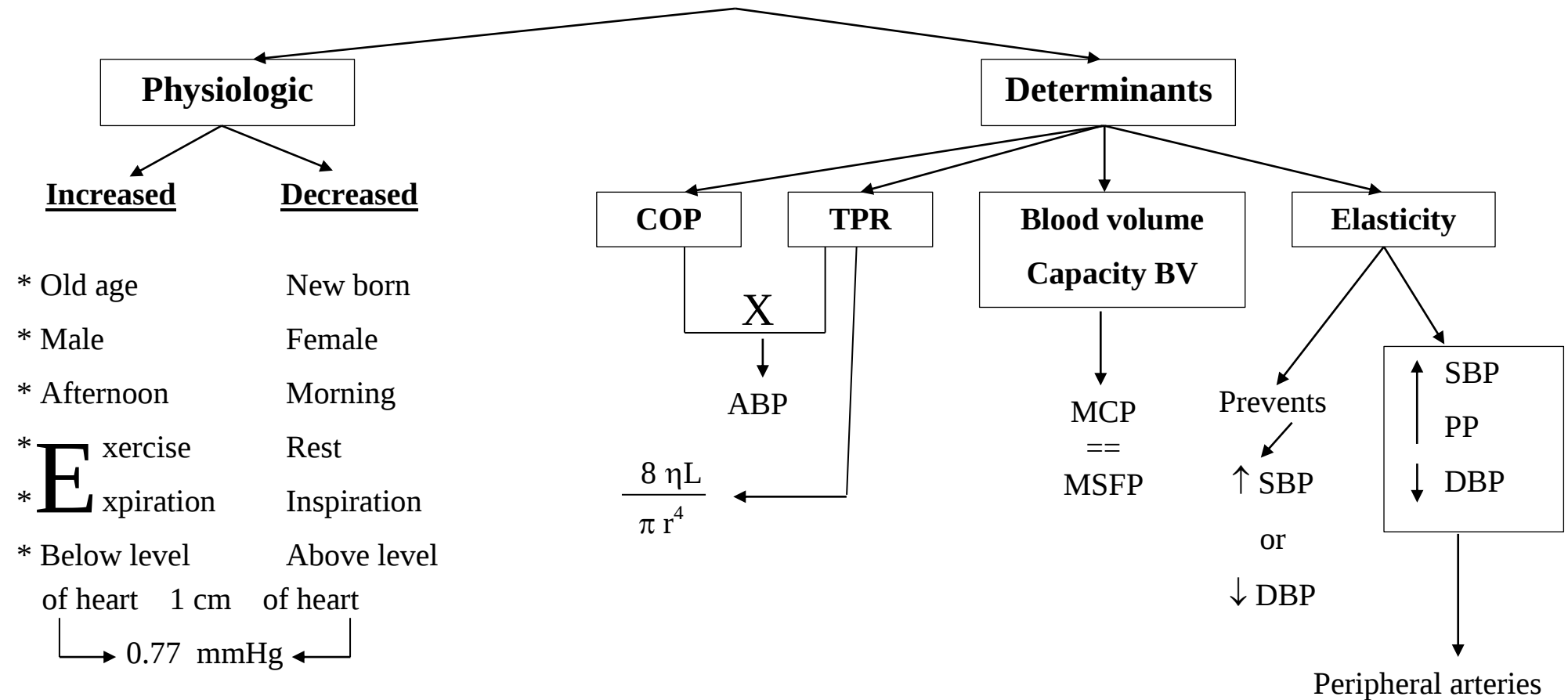
	Systolic (S)	Diastolic (D)	Pulse pressure (PP)	Mean arterial pressure (MAP)	<u>Proof: MAP = D + 1/3 PP</u>
Def.	Highest pressure on Arterial wall (Cardiac Cycle)	Lowest pressure on Arterial wall (Cardiac cycle)	S-D	Average pressure in Cardiac cycle	O. cycle S _ _ D _ _ D $MAP = (S + D + D)/3$ $\Delta PP = S - D$ $\therefore S = PP + D$
Normal	100-140 mmHg	60-90 mmHg	30-50 mmHg	93 mmHg	$\therefore MAP = (PP + D + D + D)/3$ $= D + 1/3 PP$
Average	120 mmHg	80 mmHg	40 mmHg`	90-95 mmHg	



هم MCQ

$$\frac{S + D}{2} \quad \text{MAP لا يساوي}$$

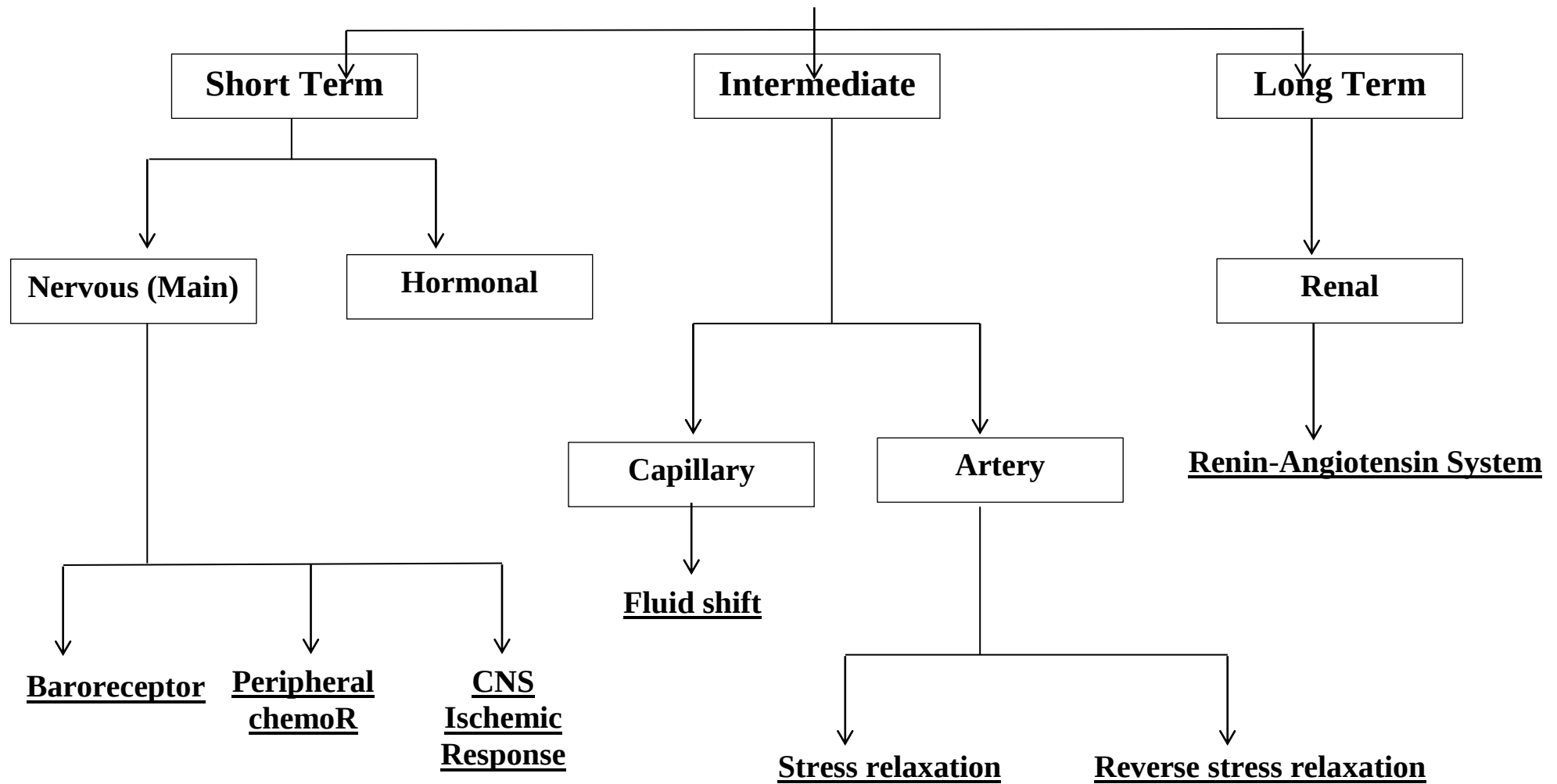
د. محمد فايز ABP



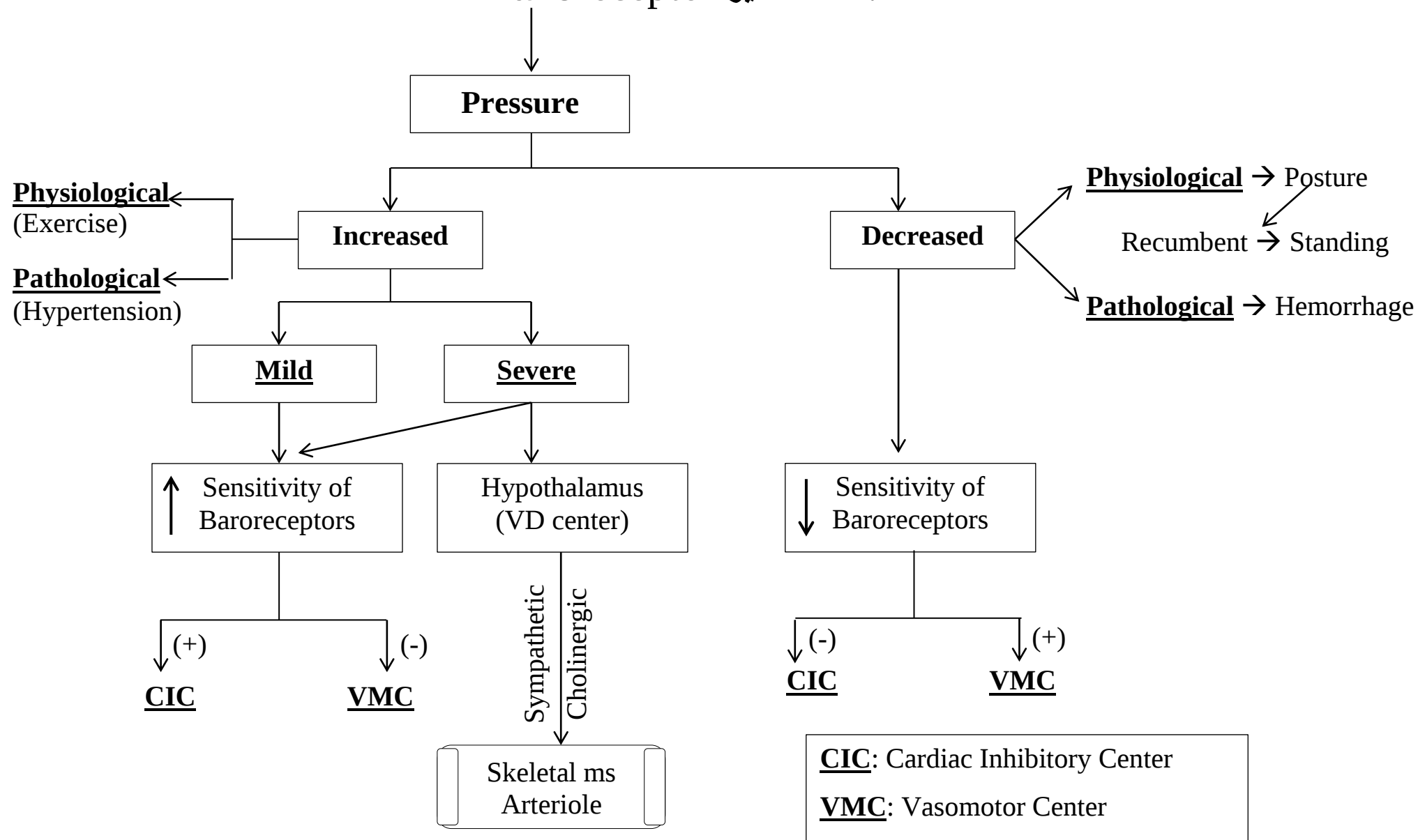
Inspiration: ↑ capacity of pulmonary vessels →
 ↓ VR (LA) → ↓ EDV → ↓ SV → ↓ COP → ↓ BP

Atherosclerosis: ↑ PP (**Water Hammer Pulse**)

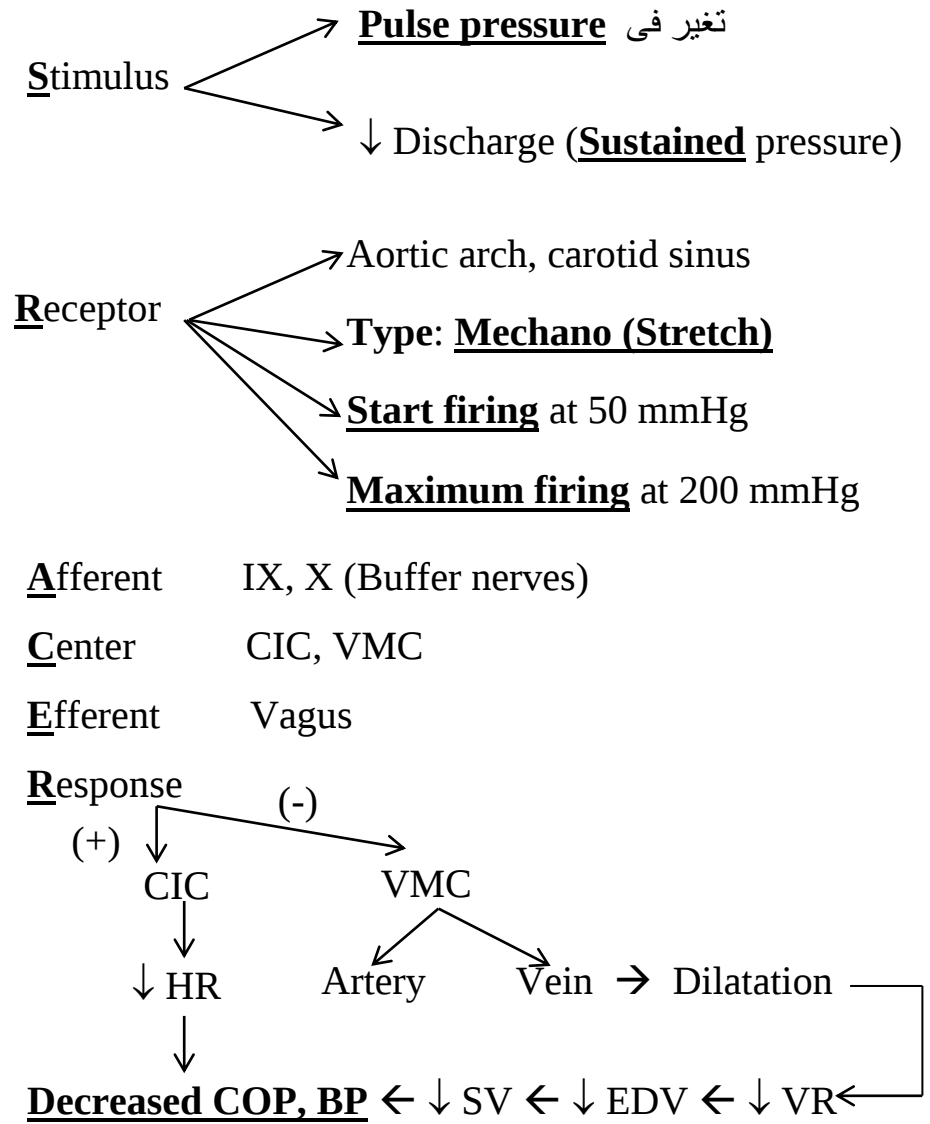
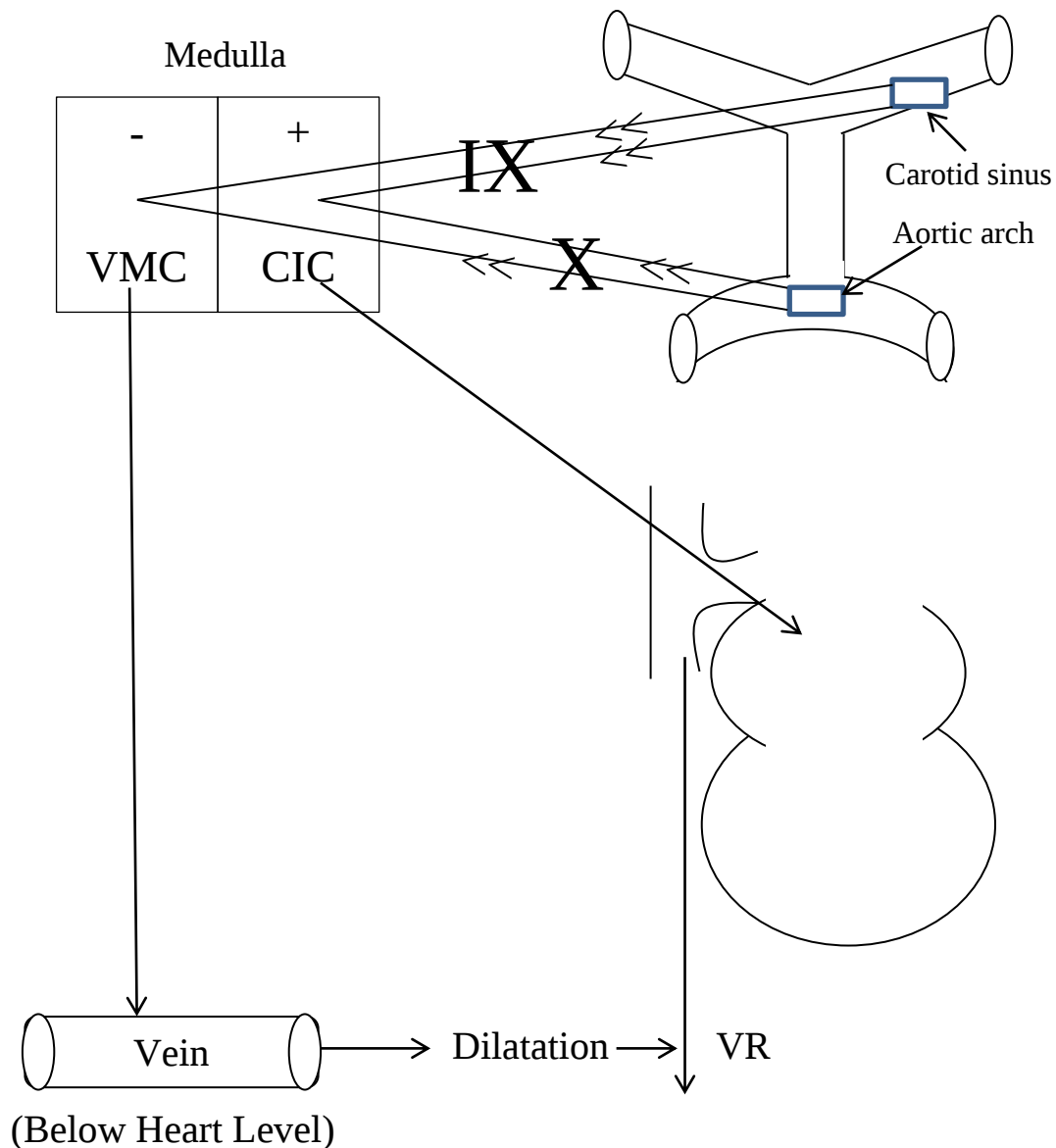
Regulation of Blood Pressure د. محمد فايز



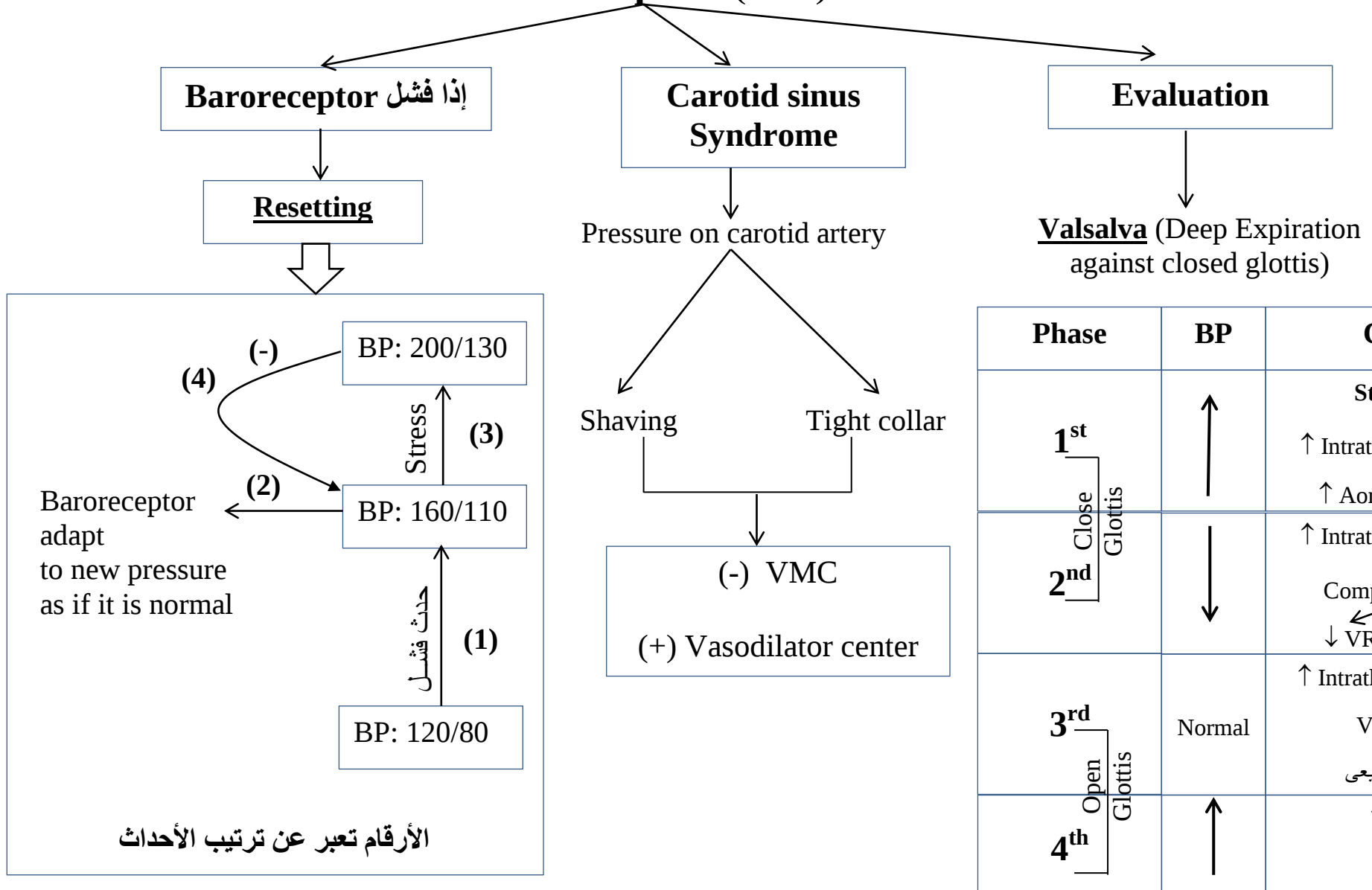
د. محمد فايز Baroreceptor



Baroreceptor Reflex د. محمد فايز



د. محمد فايز (تكملة) Baroreceptor



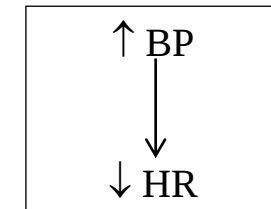
د. محمد فايز (تكملة 2) Baroreceptor

Other Types

	Atrial Receptors		Ventricular Receptors	
	High Pressure (A)	Low Pressure (B) volume	High Pressure	Bezold-Jarisch reflex
Stimulus	Atrial Systole	$\uparrow$ VR $\uparrow$ Blood volume	Ventricular Systole	* MI * Injection of Veratridine Serotonin Nicotine
Response	VD $\downarrow$ HR $\downarrow$ Contractility	* Reflex VD $\downarrow$ $\uparrow$ capillary Pr. $\downarrow$ Capillary fluid shift (Intermediate) * $\downarrow$ ADH, $\uparrow$ ANP $\uparrow$ urine (Long Term)	VD $\downarrow$ HR $\downarrow$ Contractility	$\downarrow$ HR $\downarrow$ BP

Effect on Respiratory Center

Adrenaline Apnea أنظر ص

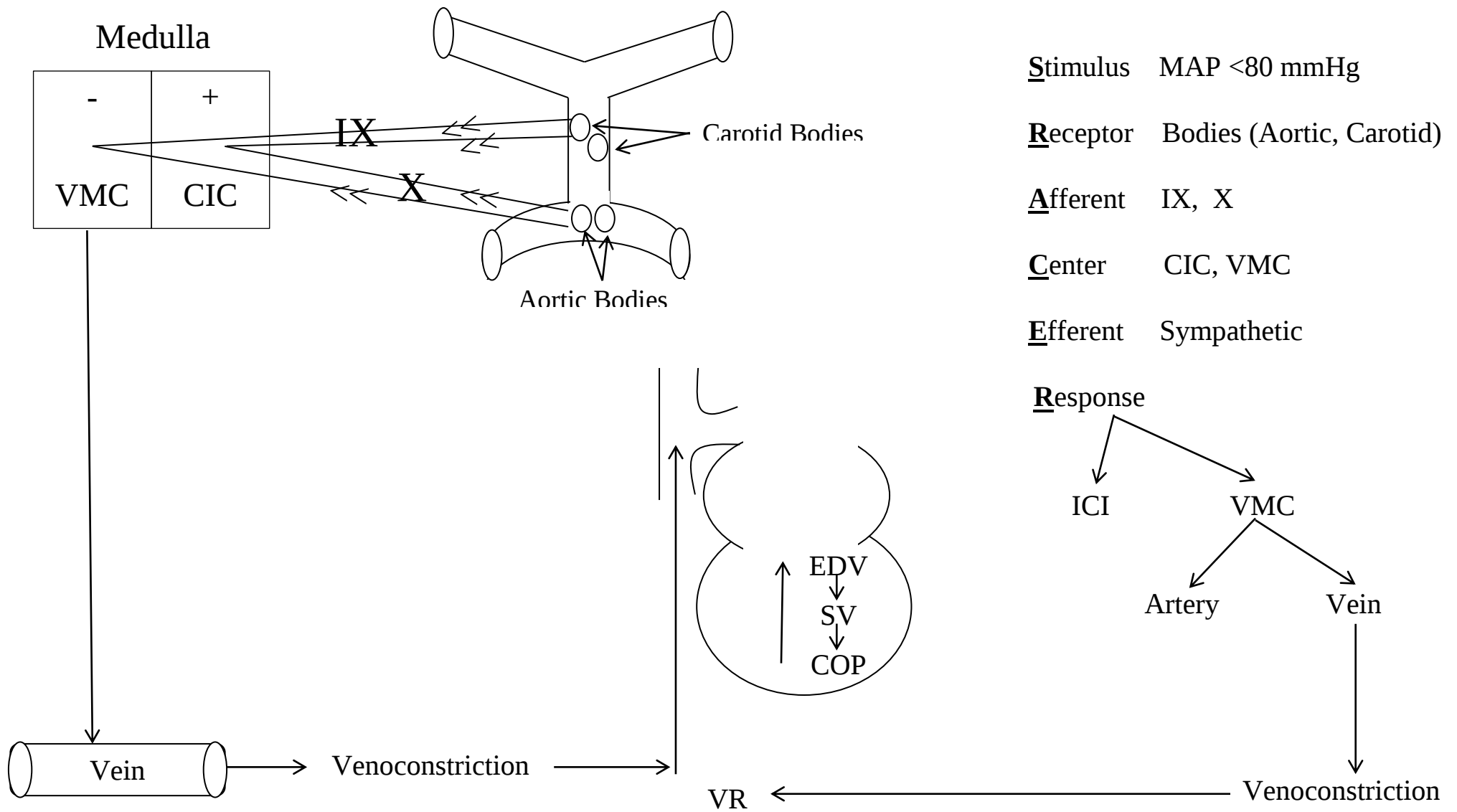


Marys' Law

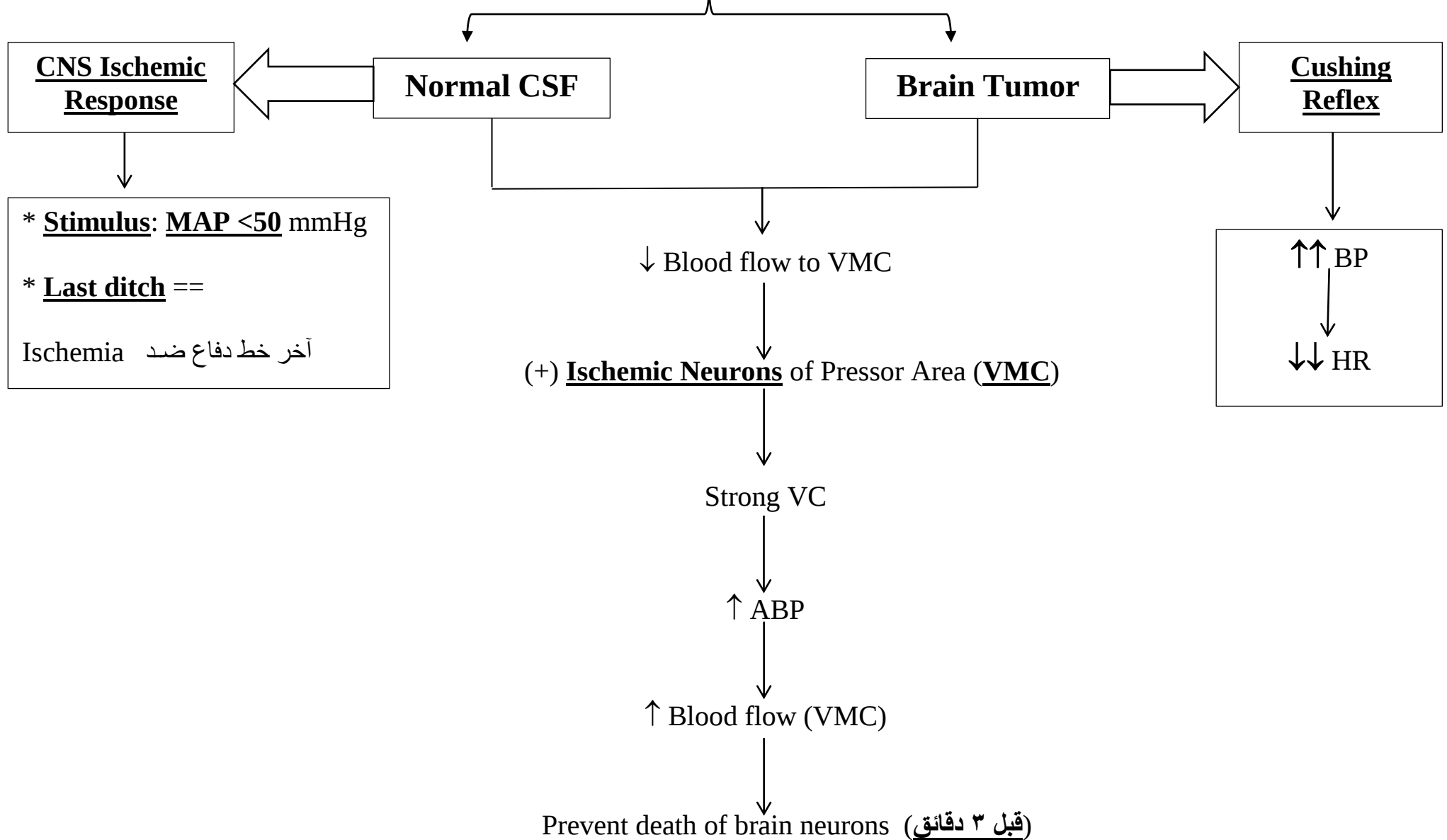
لا يوجد Cardiac Stimulatory Center

Adrenaline Apnea $\rightarrow$ Low Pressure (Act as Atrial Type B)
 $\rightarrow$ High Pressure

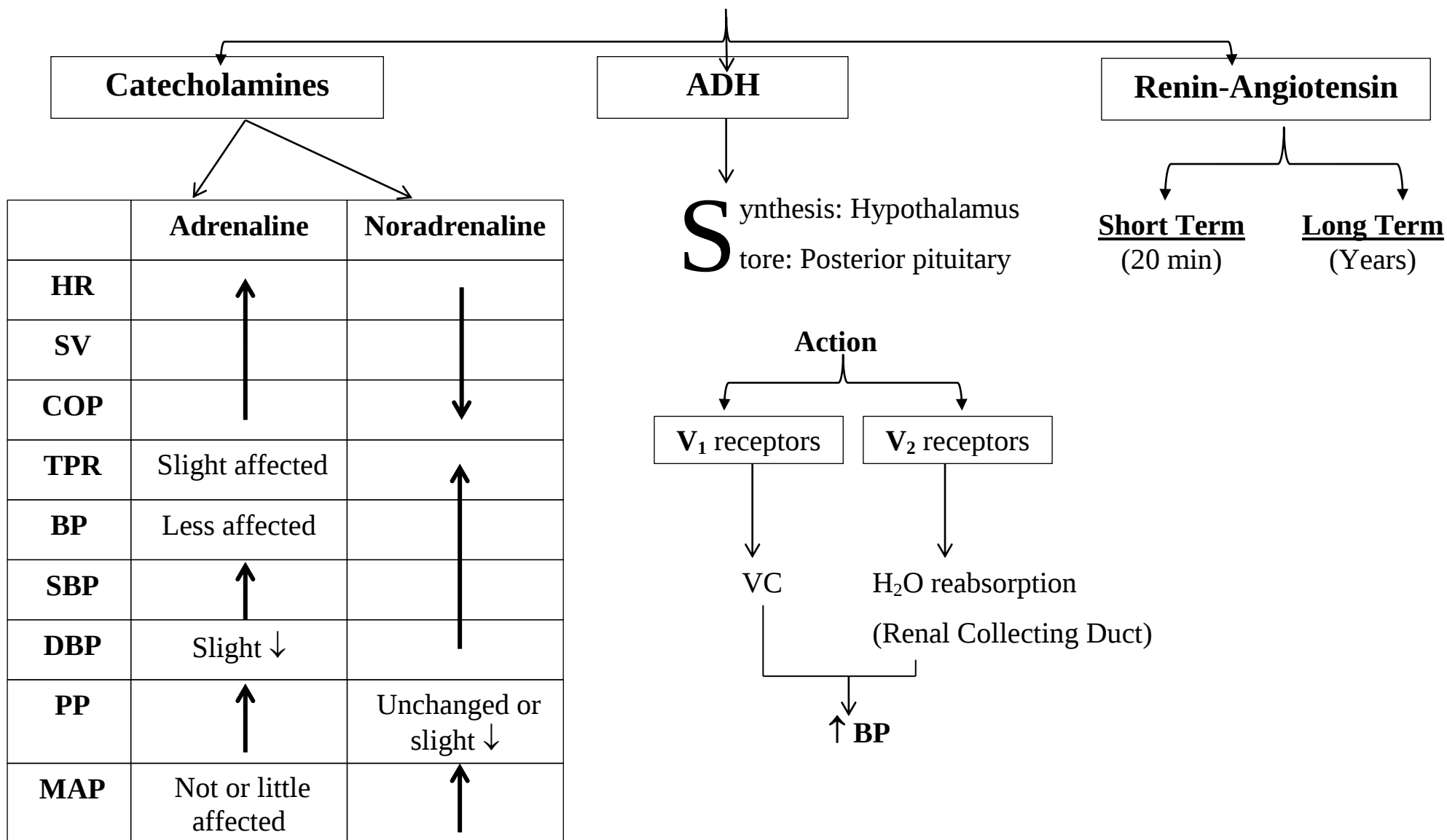
د. محمد فايز Peripheral Chemoreceptor Reflex



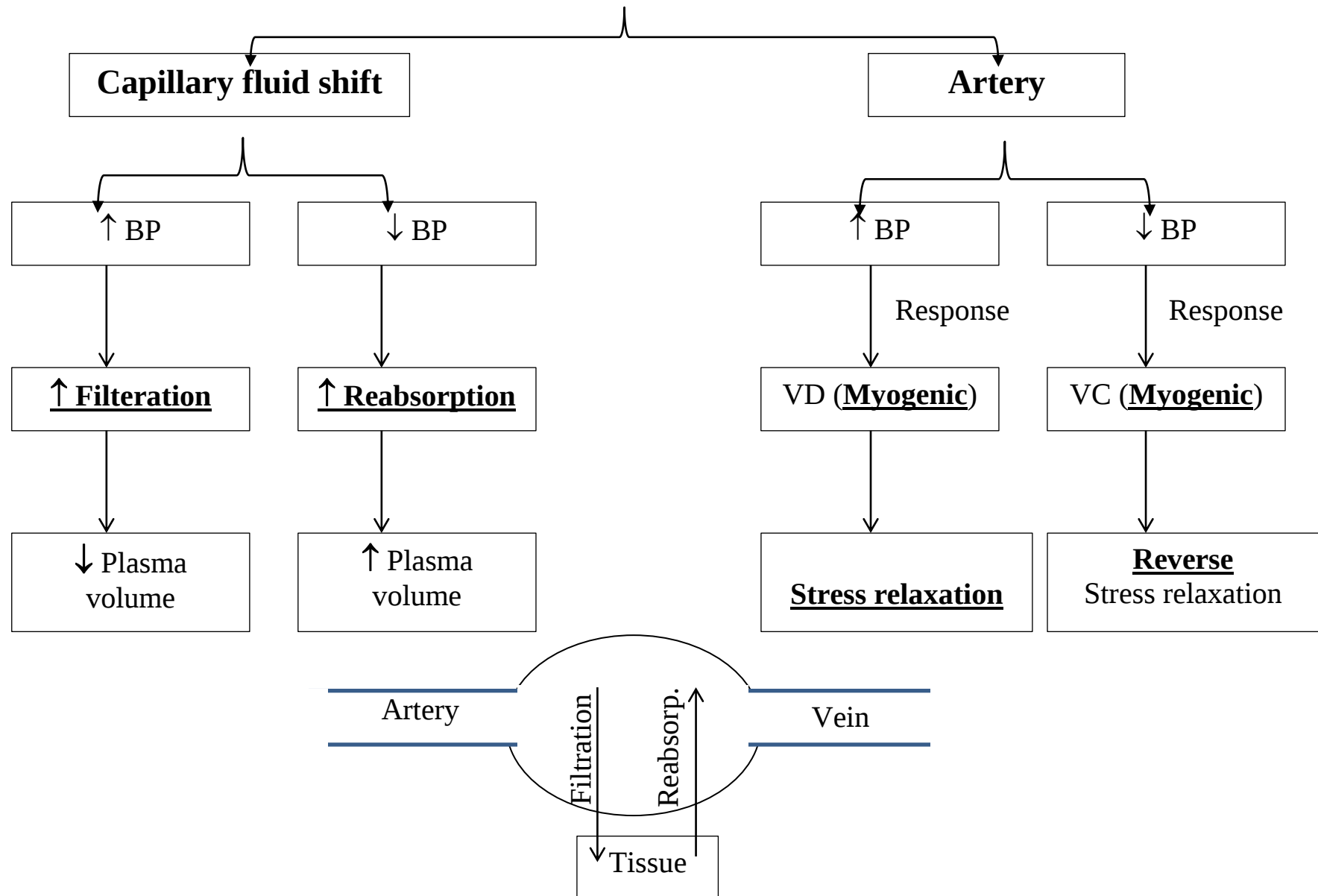
د. محمد فايز Cerebral Ischemia



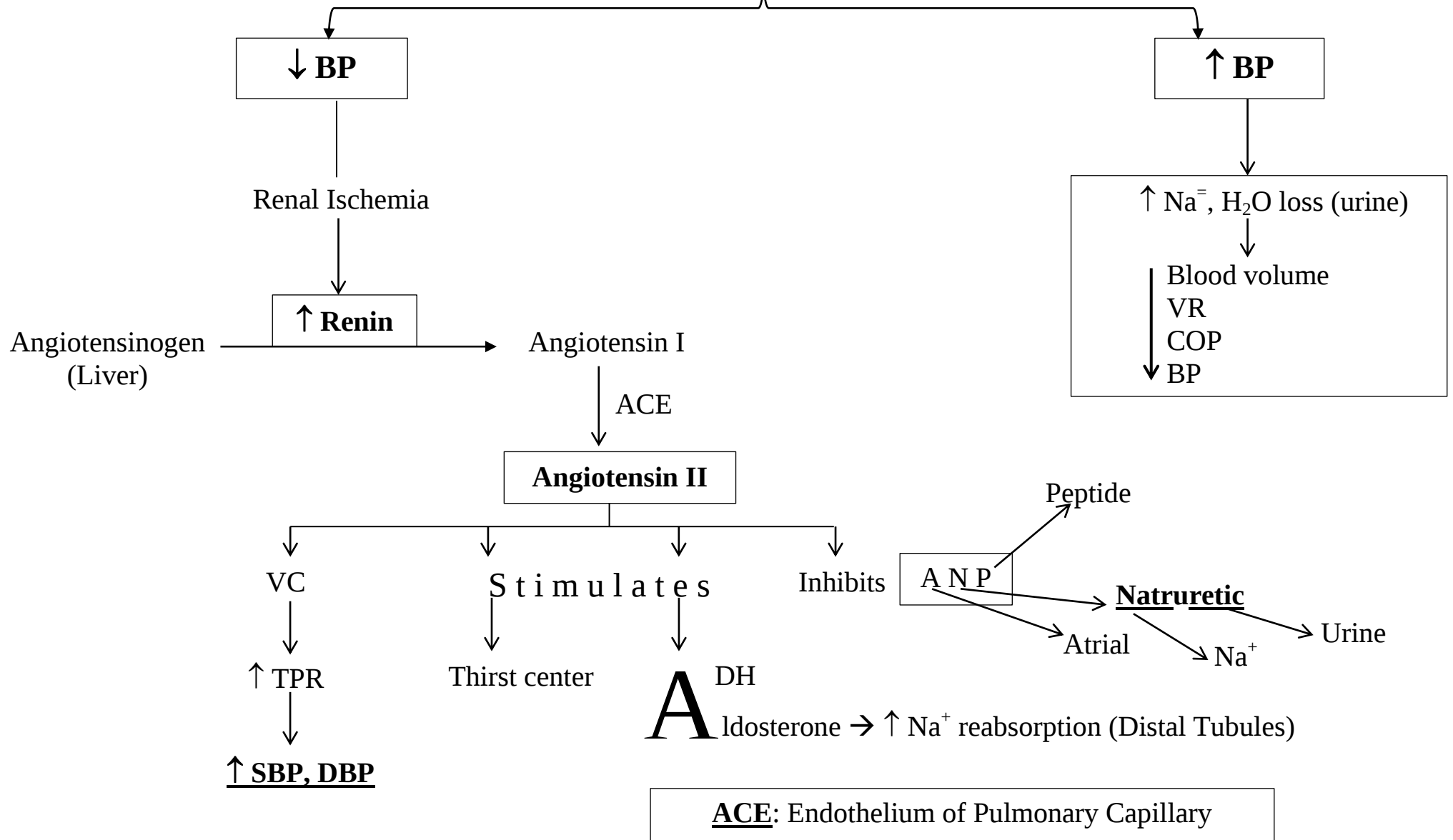
د. محمد فايز Rapidly Acting Hormones



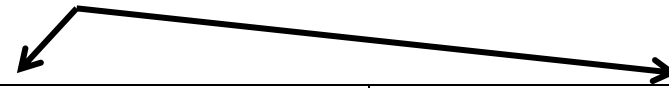
د. محمد فايز Intermediate Regulation of ABP



د. محمد فايز Long term regulation (Renal)

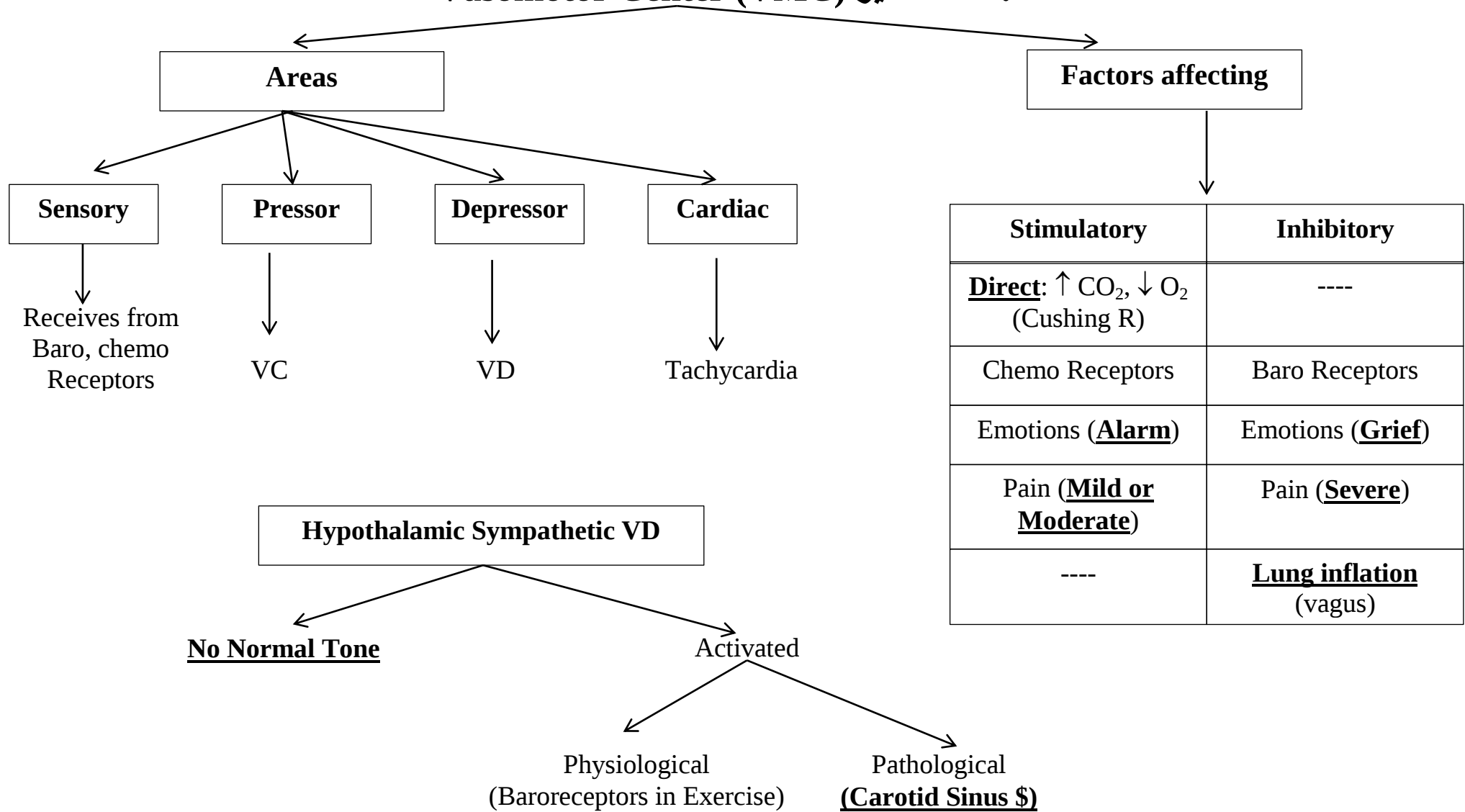


د. محمد فايز Compare Regulation of BP

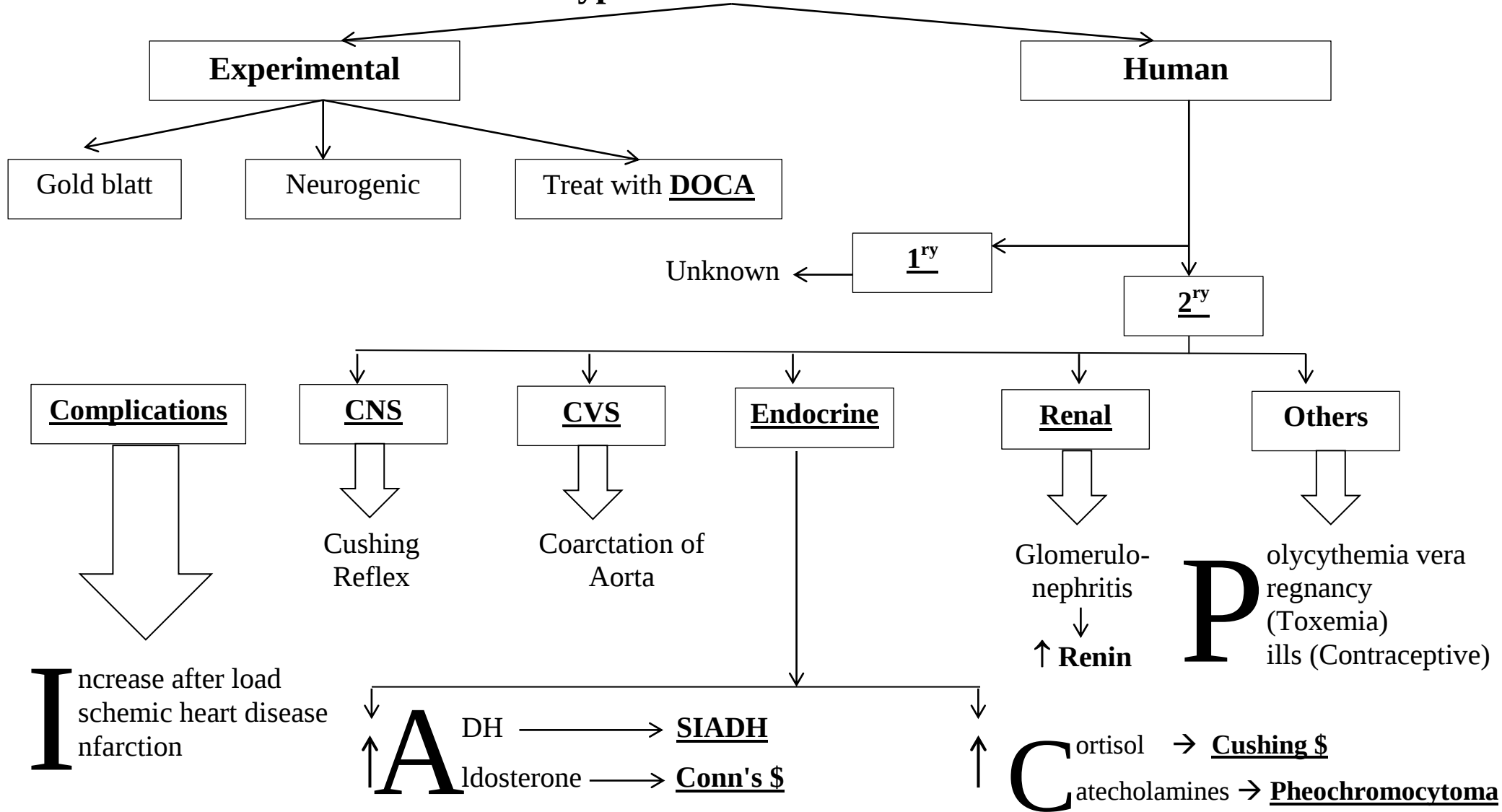


	Shot-Term	Long-Term
	<u>Rapid</u>	<u>Slow</u>
Act	Seconds or Minutes	Hours or Days
Control BP	<u>Moment to Moment</u>	Days/Weeks/Months
Regulate BP	Change <u>Capacity</u> of BV	Change <u>ECF Volume</u>
Mainly	<u>Nervous</u>	Renal, Hormonal
Effectiveness	↓ (Adaptation)	↑
Potency	<u>Moderate</u>	<u>Extreme</u>

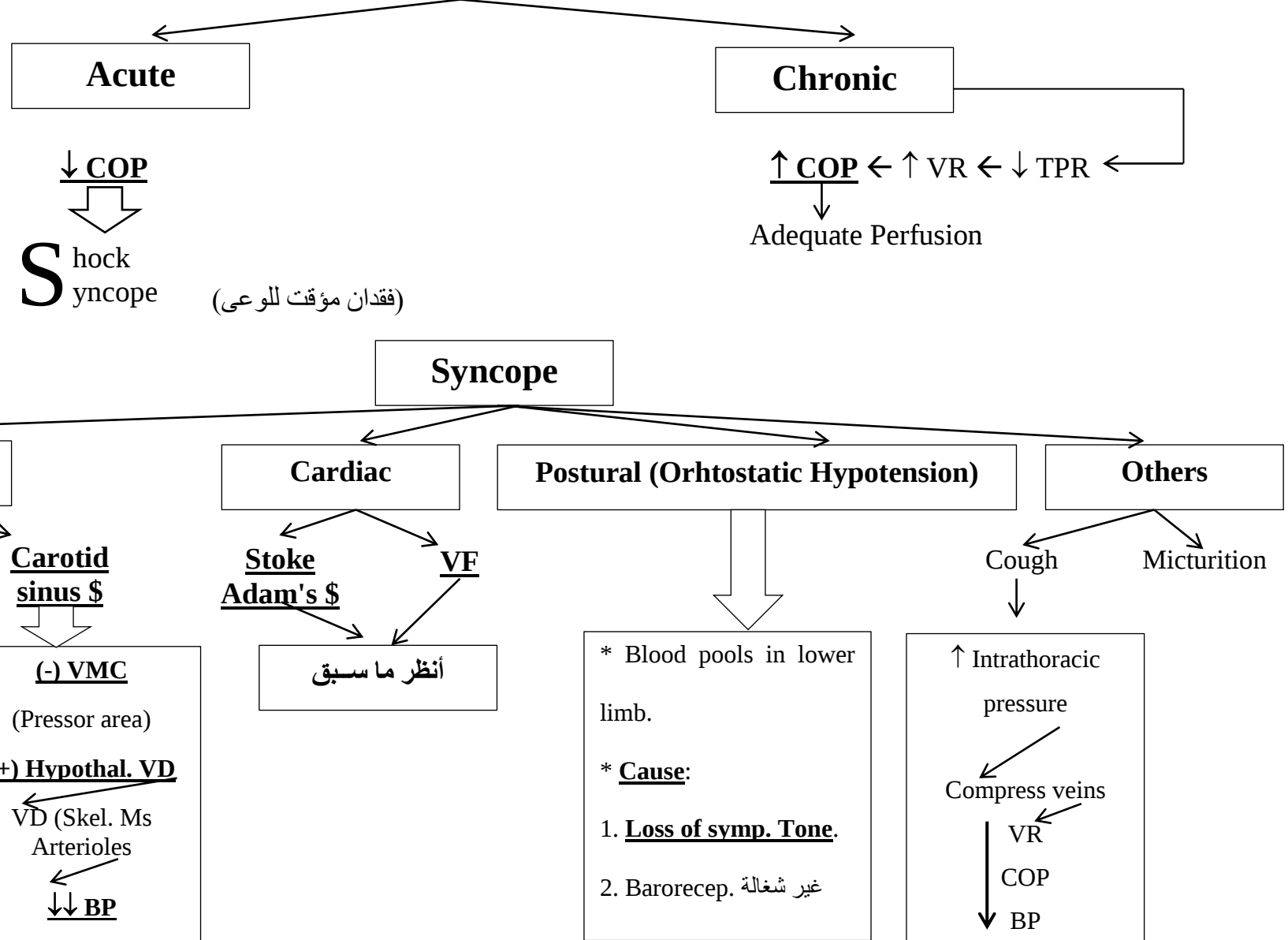
د. محمد فايز Vasomotor Center (VMC)



د. محمد فايز Hypertension



د. محمد فايز Hypotension

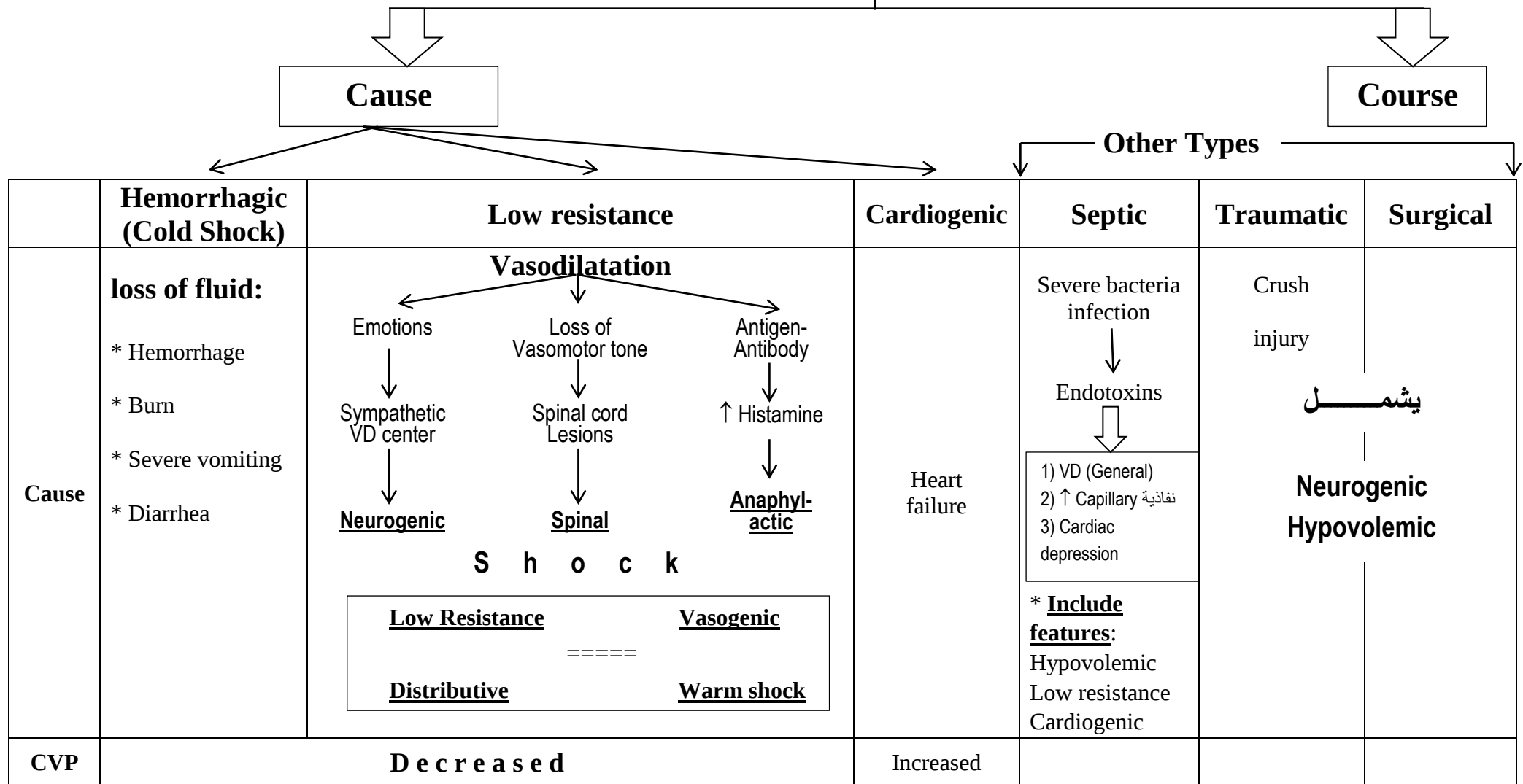


د. محمد فايز Hemorrhage

Compensatory Reactions

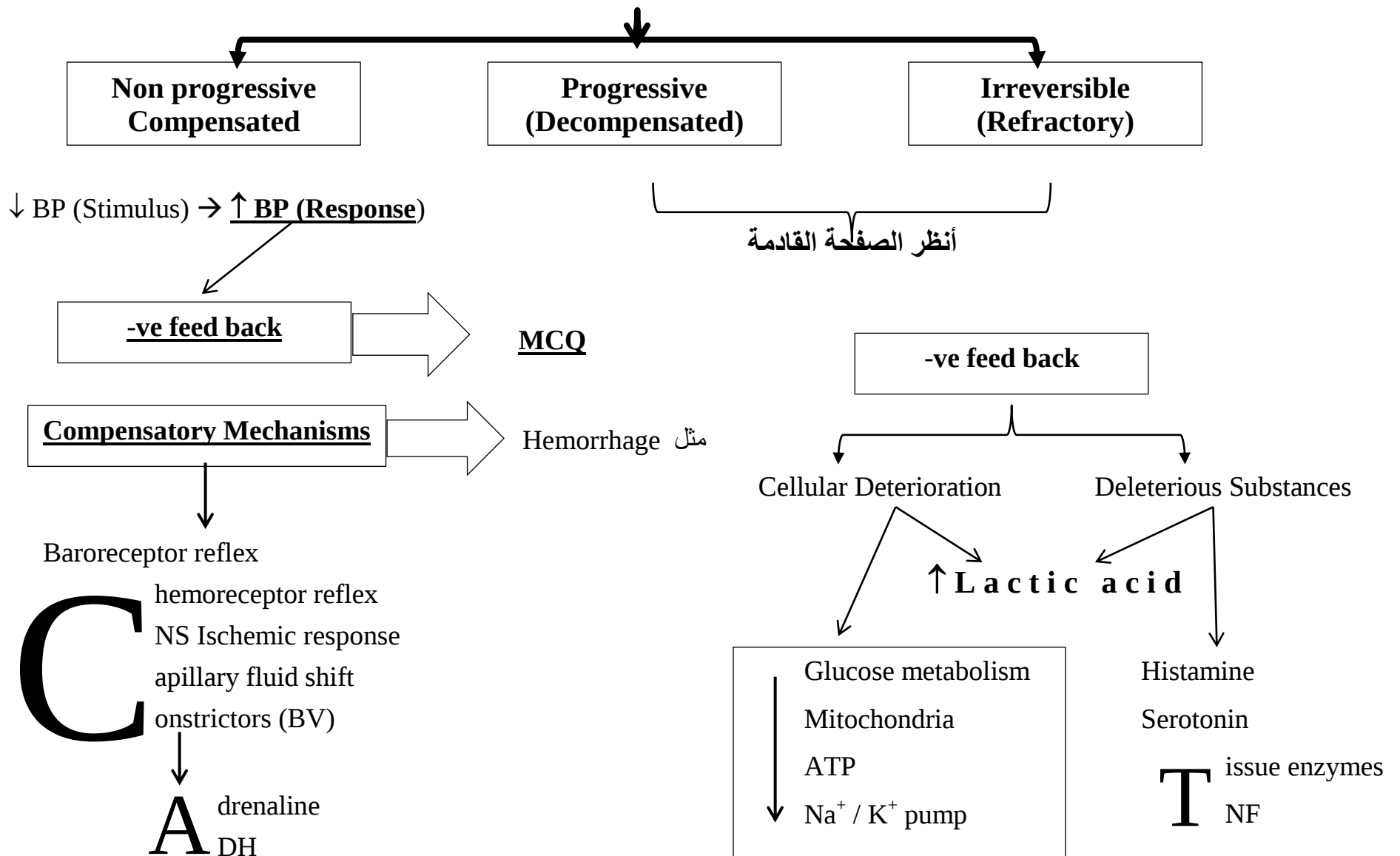
	Short Term (Immediate)	Long Term
Correct ↓ BP	1) ↓ <u>Baroreceptor discharge</u> (+) <u>VMC</u> Venoconstrict VC VR EDV SV COP <u>Arterioles</u> → ↑ TPR <u>Skin</u> → Pale, cold <u>Renal</u> → ↓↓ urine vol. Sympathetic Heart Adr. Med. ↑ HR ↑ Catechol. Rapid, weak pulse Restless Compensatory VC → ↑ DBP → ↓ Pulse Pressure 2) ↑ <u>Peripheral chemoreceptor reflex</u> (إشرح) 3) <u>Capillary fluid shift</u> (إشرح) 4) <u>Reverse stress relaxation</u> (إشرح) 5) ↑ <u>Angiotensin II, ADH</u> (إشرح)	إشرح Renin-Angiotensin System
Correct ↓ Volume	<u>Plasma volume</u> restored by <u>capillary fluid shift</u> <u>Plasma proteins</u> restored by <u>moving liver proteins to plasma</u> <u>RBCs restored</u> by <u>spleen contraction</u>	Plasma vol. restored (½-3 days) by Thirst, ADH Plasma ptn restored (3-4 dys) by hepatic synthesis RBCs restored (4-8 weeks) by ↑ erythropoietin

د. محمد فايز Shock (Tissue Underperfusion)

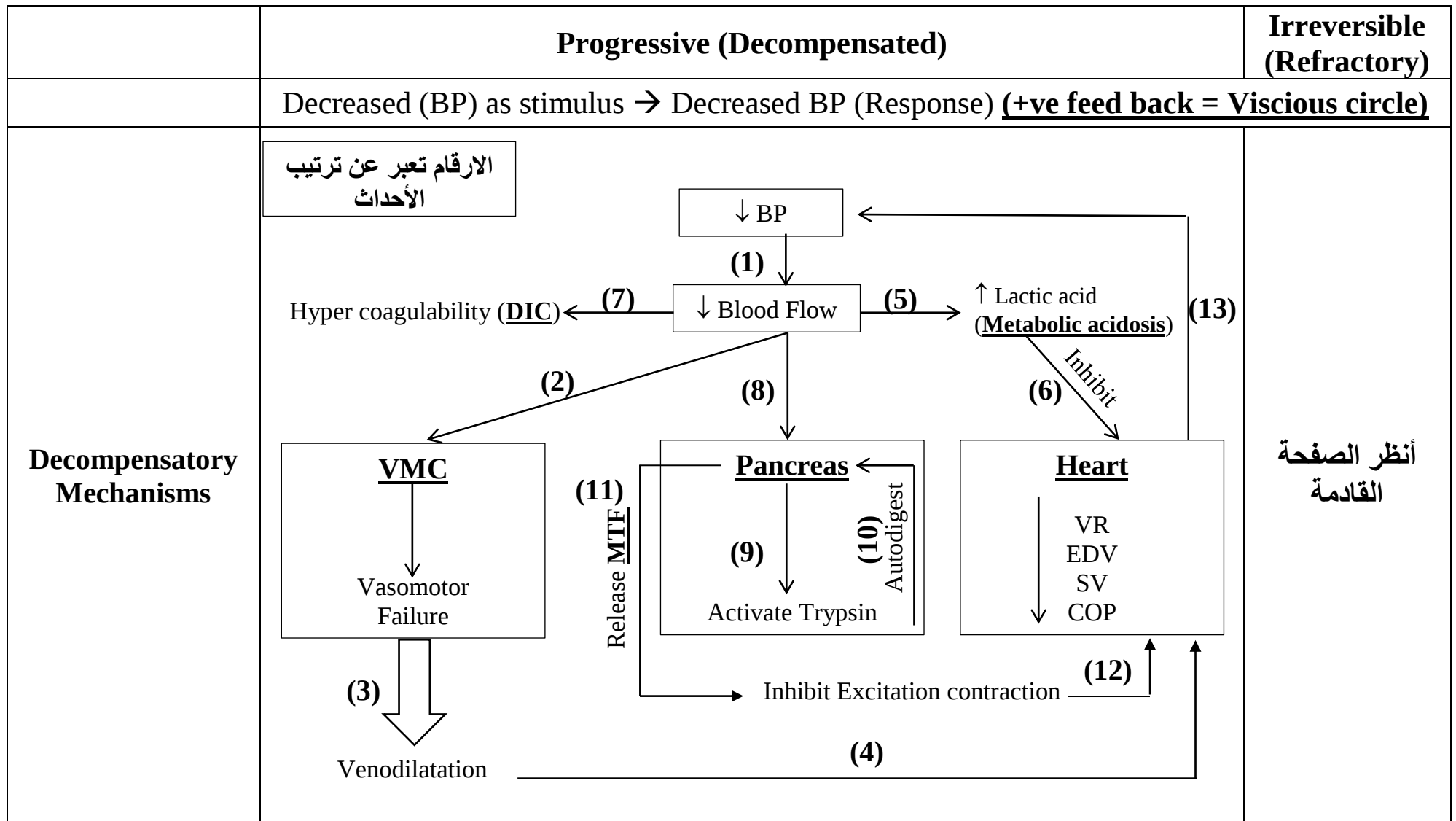


N.B.: ABP not a good measure for progression of shock.

Course of Shock د. محمد فايز



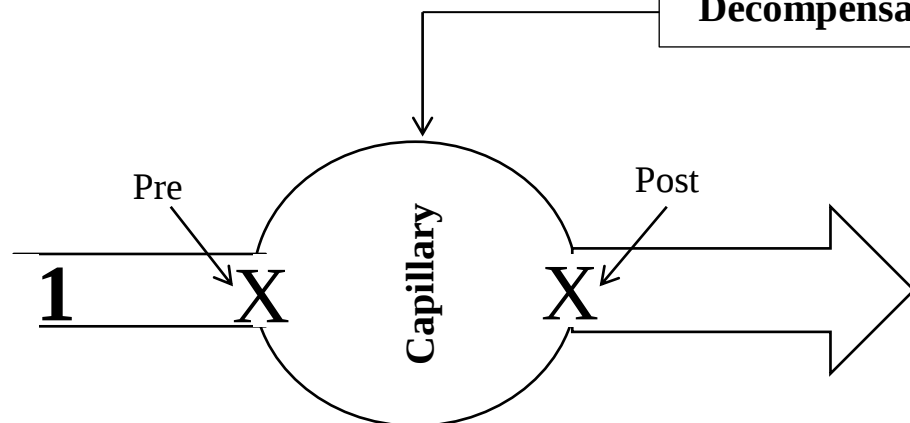
د. محمد فايز Course of Shock



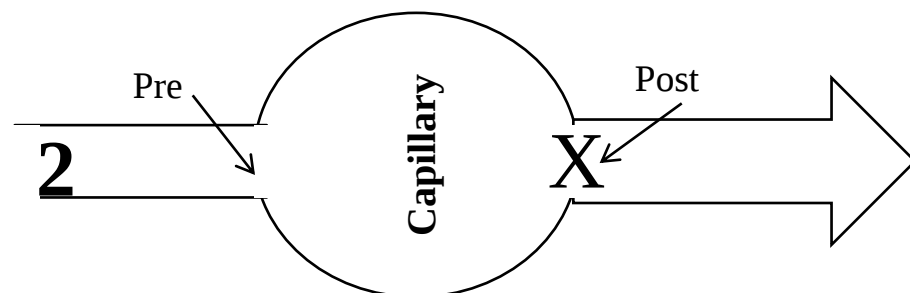
د. محمد فايز Irreversible (Refractory Shock)



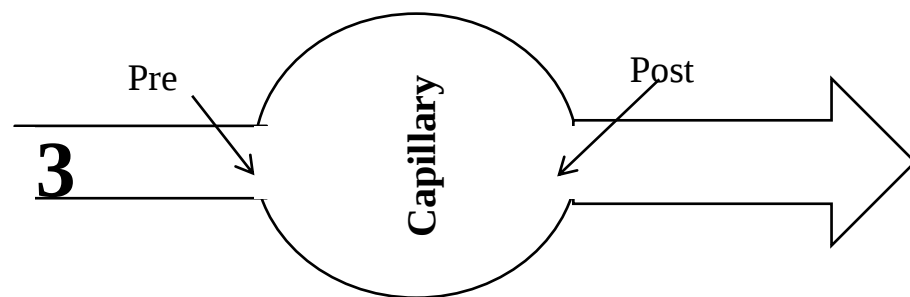
Decompensatory Mechanisms



Sympathetic constricts (pre, post capillary sphincters) in
(Hypovolemic and Cardiogenic shock)

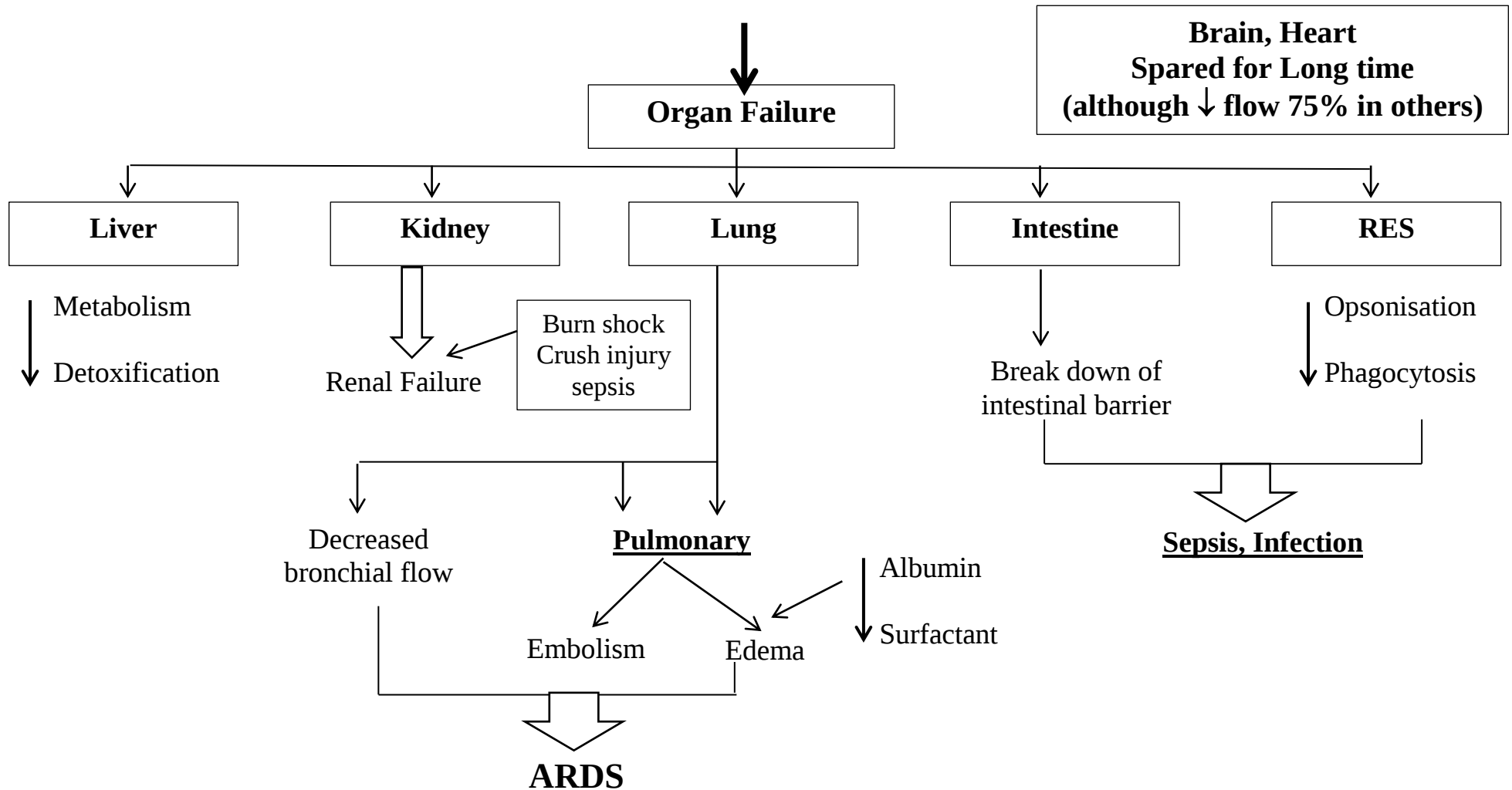


Severe local hypoxia → Accumulation of VD substances
→ Dilate precapillary sphincter (Post capillary closed)



Post capillary sphincter relax → Packed RBCs and small
fragmented clots reach circulation → Pulmonary embolism

د. محمد فايز Course of Shock



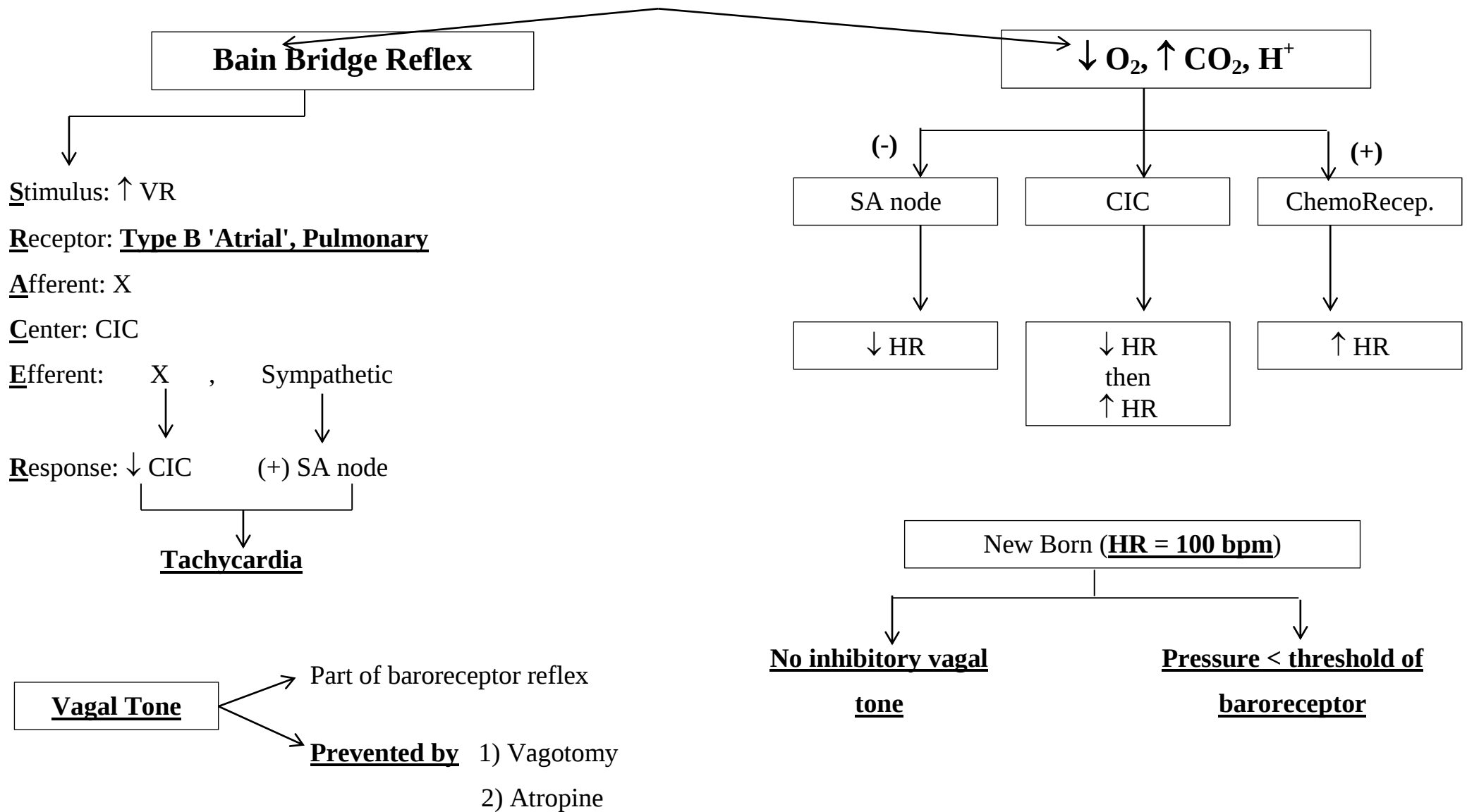
د. محمد فايز Factors Affecting HR



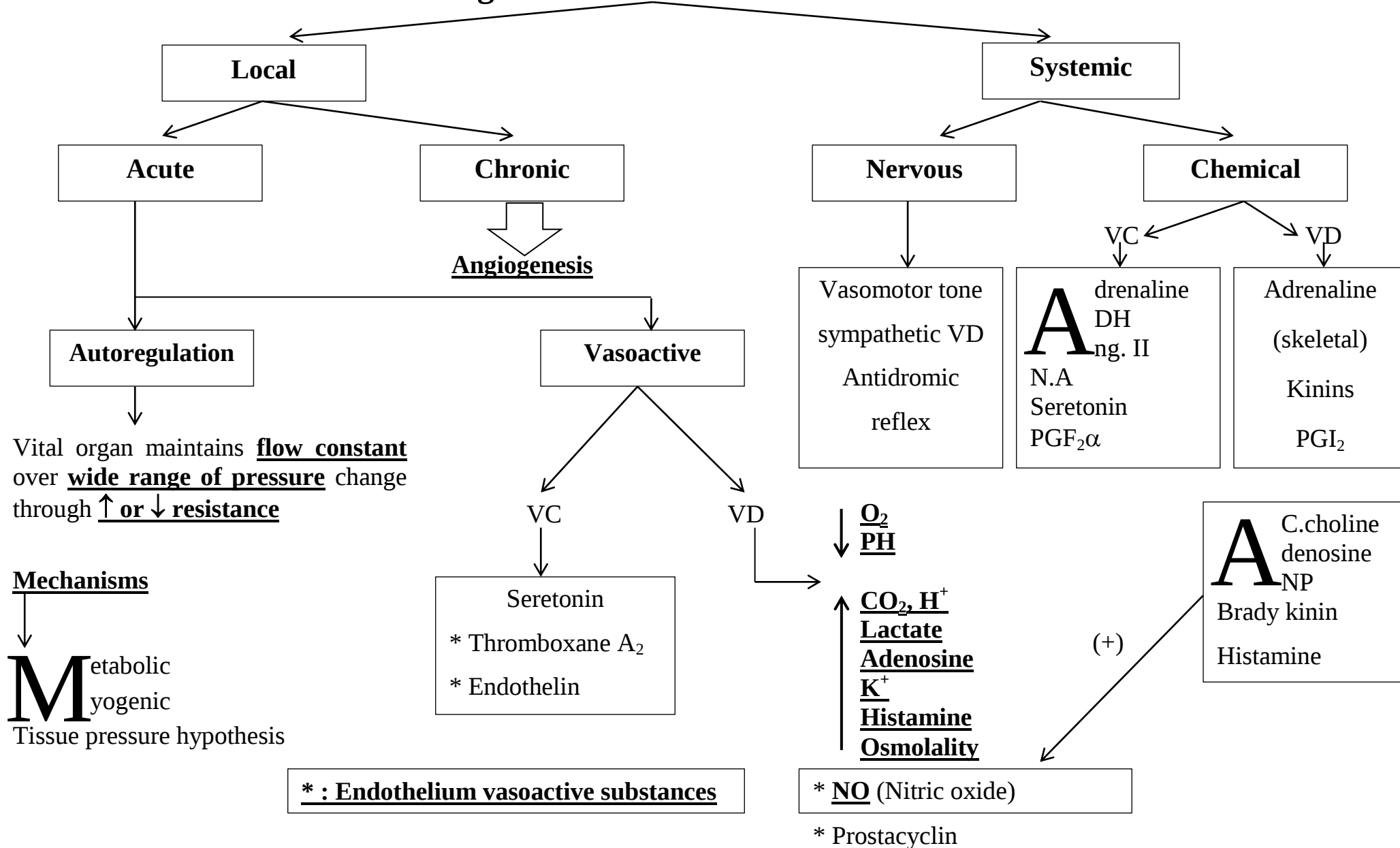
	Tachycardia	Bradycardia
Higher centers	Pain (<u>Mild or Moderate</u>)	Pain (<u>Severe</u>)
cortex	Emotions (<u>Alarm reaction</u>)	Emotions (<u>Grief</u>)
Reflexes	1) chemo Receptors 2) Atrial <u>Type B</u> 3) Muscle, joint receptors	1) Baroreceptors 2) Ventricular, Atrial <u>Type A</u> 3) Cushing.
Respiration	Inspiration	Expiration
Hormonal	Adrenaline, NA, Thyroxine	--
Drugs	Thyroxine Sympathomimetics	β -Blockers Ca^{++} Blockers
Heat	↑ Temperature	Fever

هام: تأثير Adrenaline من اقوى على BP ولكن تأثير Adrenaline اقوى على HR

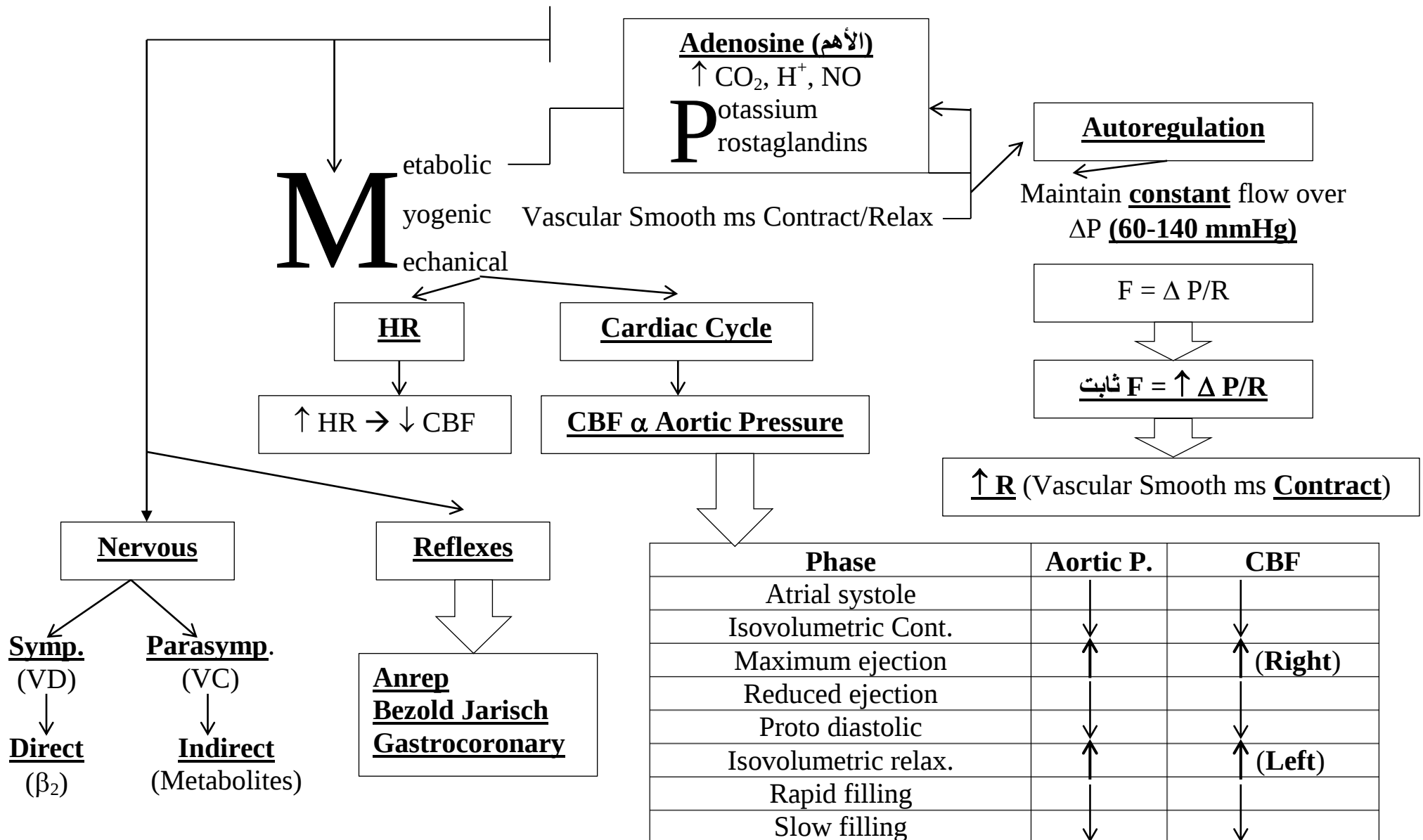
د. محمد فايز Notes on HR



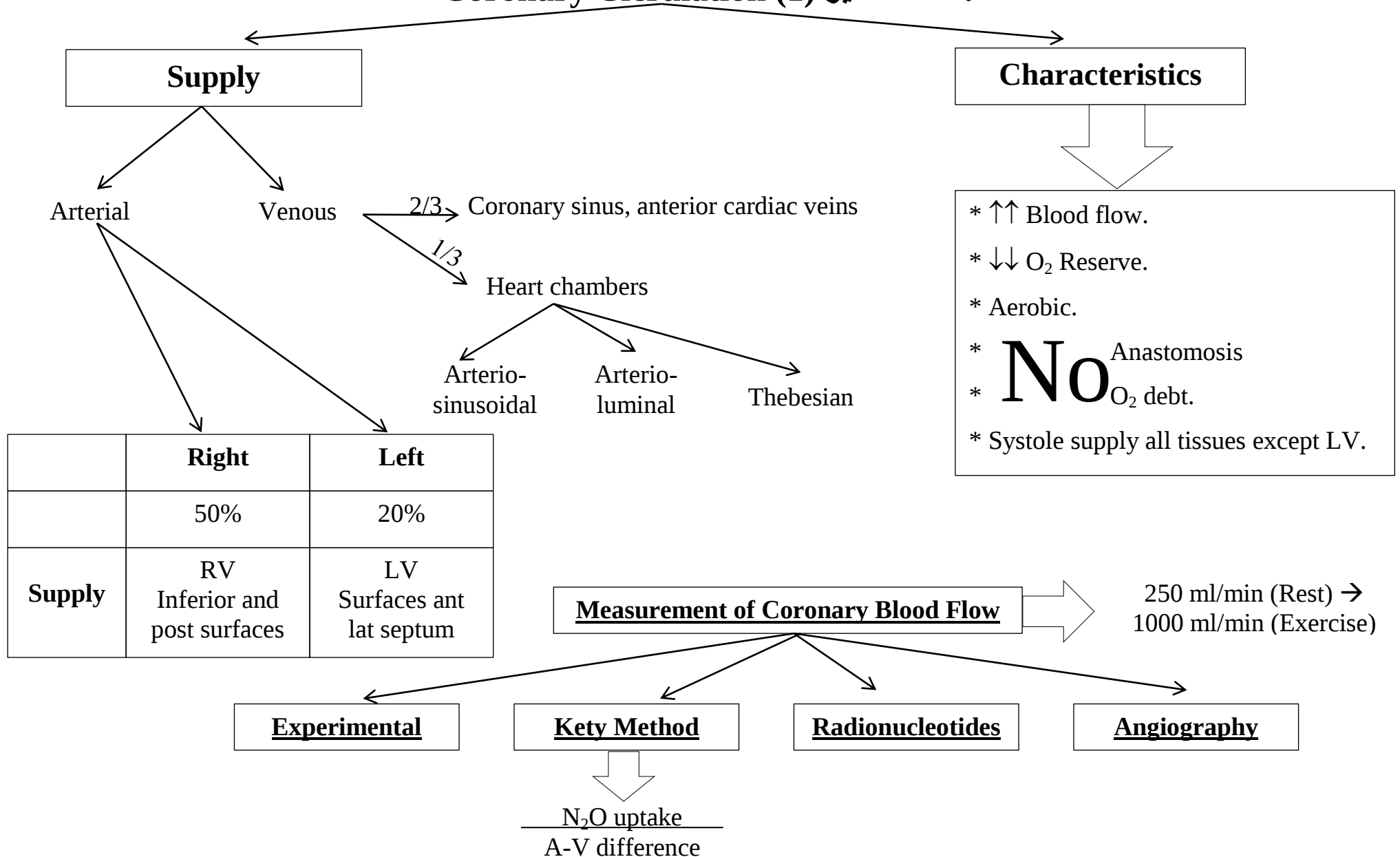
Regulation of blood flow د. محمد فايز



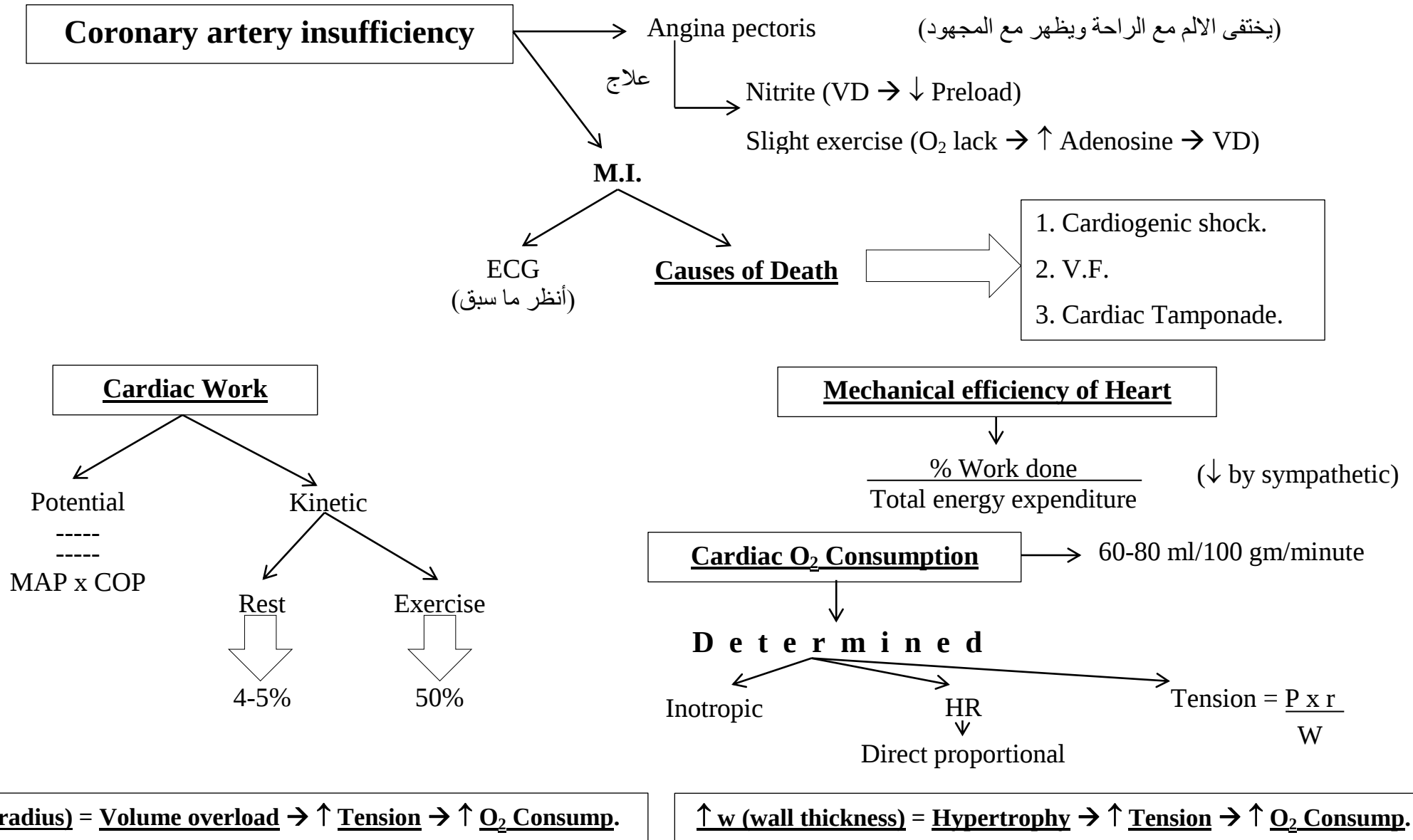
د. محمد فايز Regulation of Coronary Blood Flow (CBF)



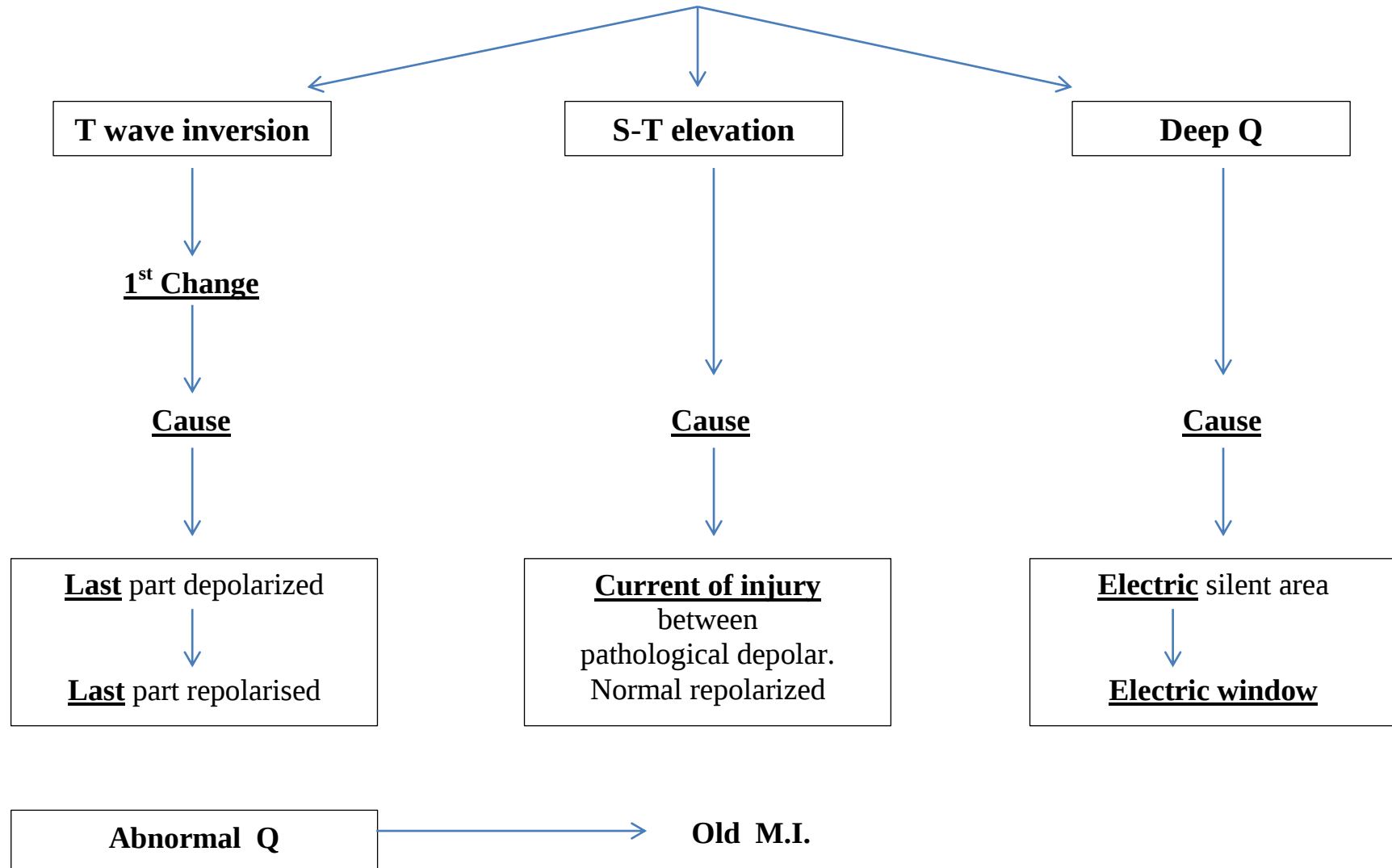
د. محمد فايز (1) Coronary Ciculation



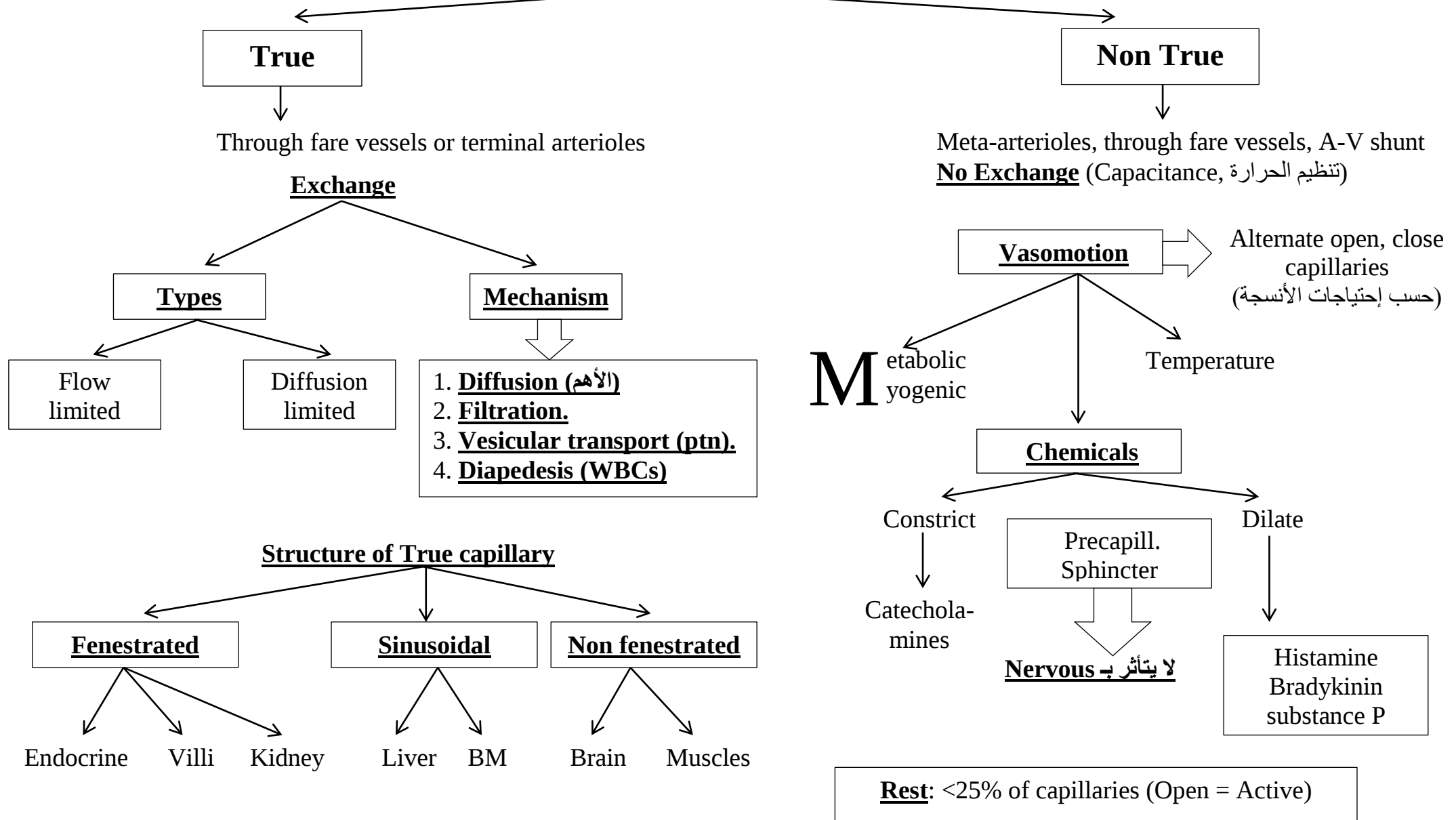
د. محمد فايز (3) Coronary Ciculation



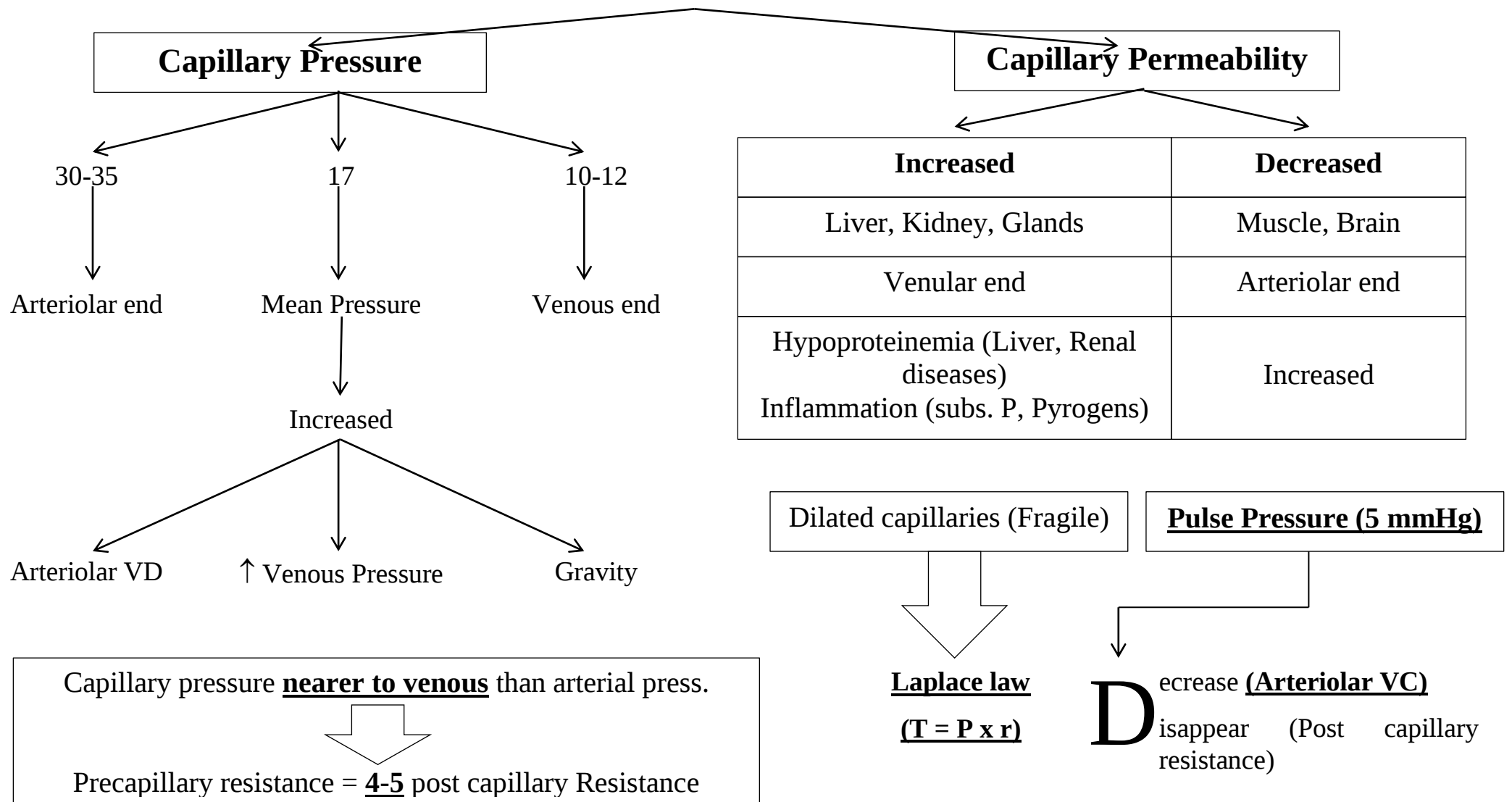
د. محمد فايز (M.I) Coronary Insufficiency



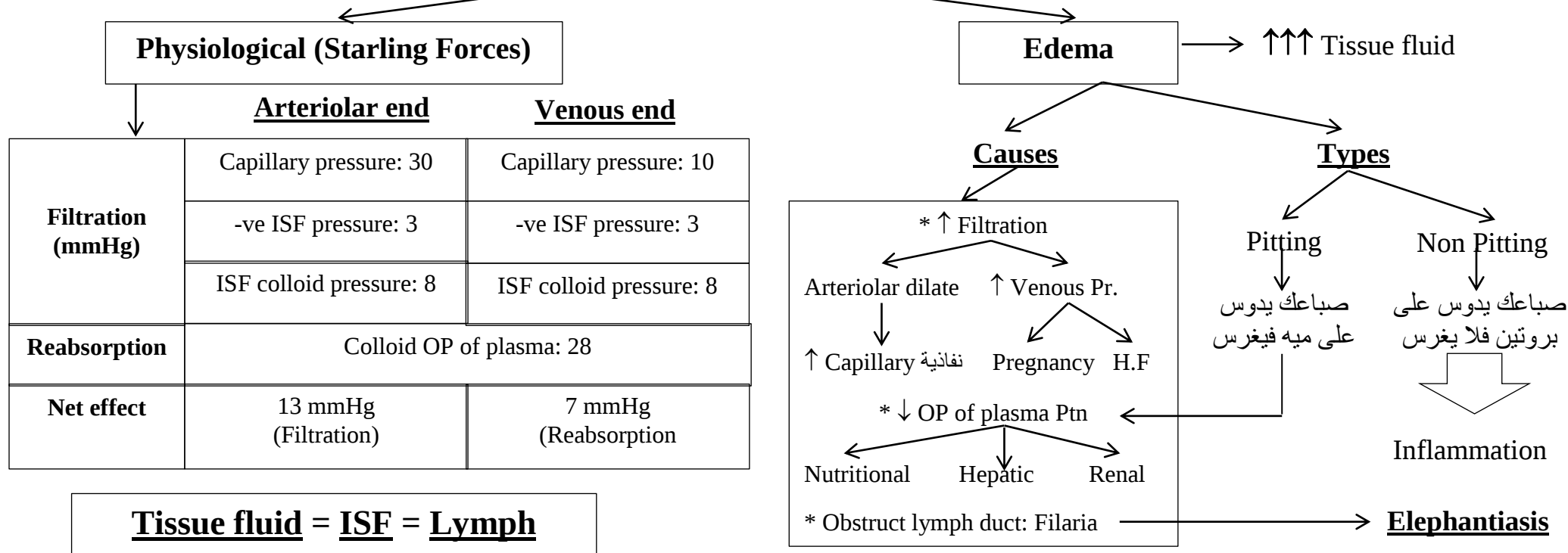
د. محمد فايز Capillary Circulation



د. محمد فايز (تكملة) Capillary Circulation



د. محمد فايز Tissue Fluid Formation



	Lymph	Plasma
Albumin	0.5-1 gm%	4.5 gm%
A/G ratio	↑	↓
Composition	N e a r e r	

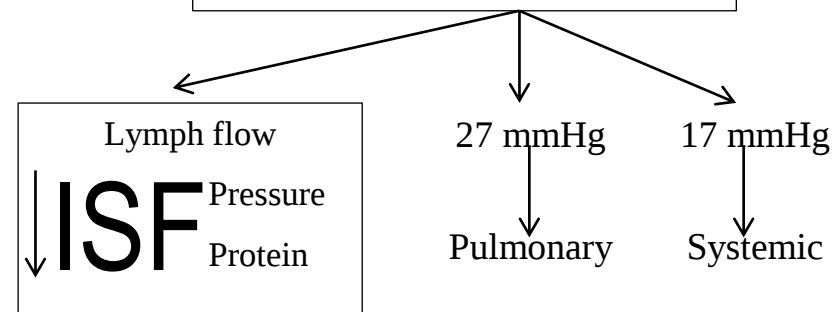
Factors which ↑ lymph flow:

Lymphatic pump (VR مثل)
 ↑ ISF pressure
 ↑ Activity of tissues

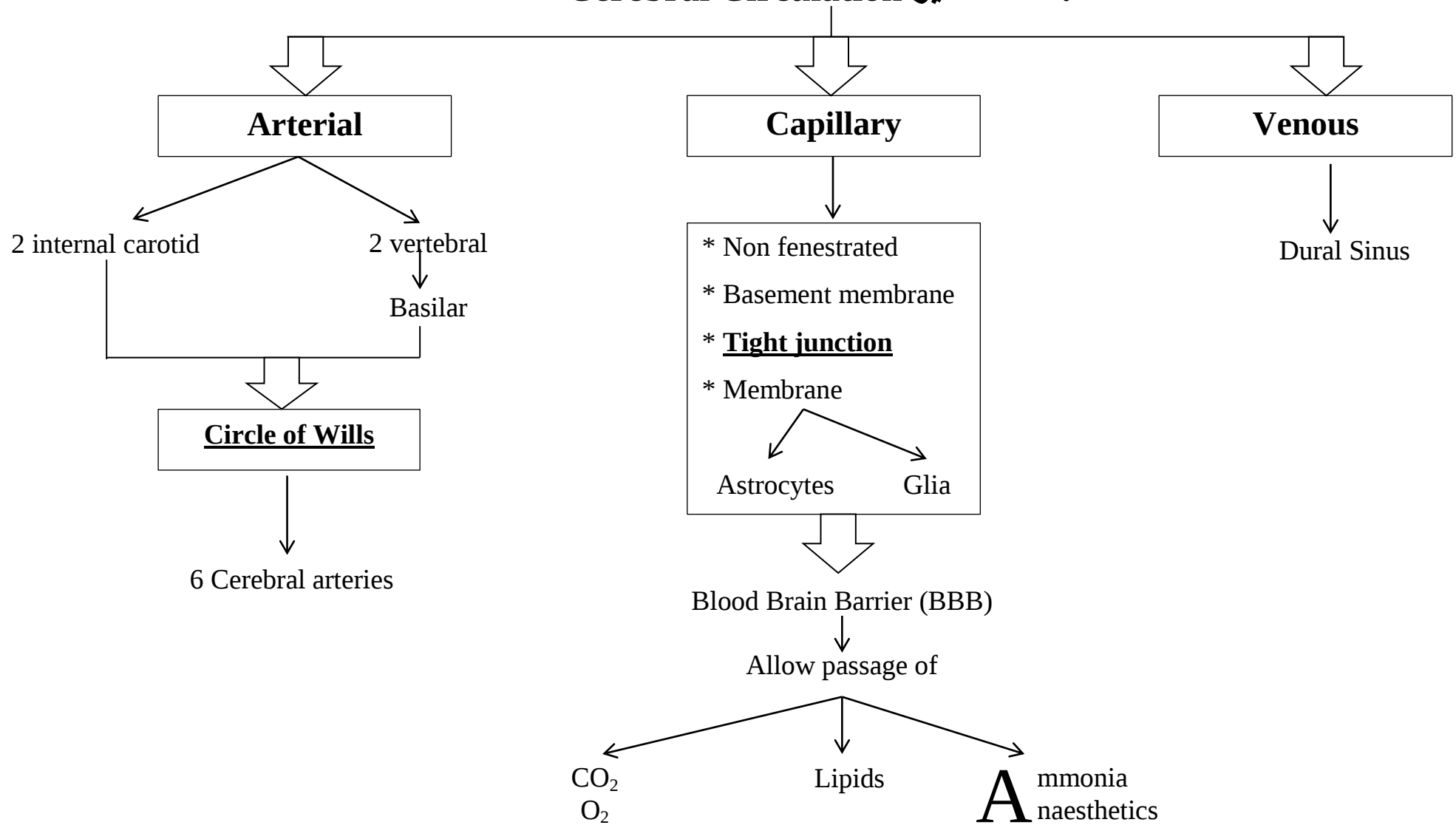
Lymphagogues (↑ Lymph)

- 1^{ry}: Bacterial Toxins, Peptones
 2^{ry}: Hypertonic Solutions

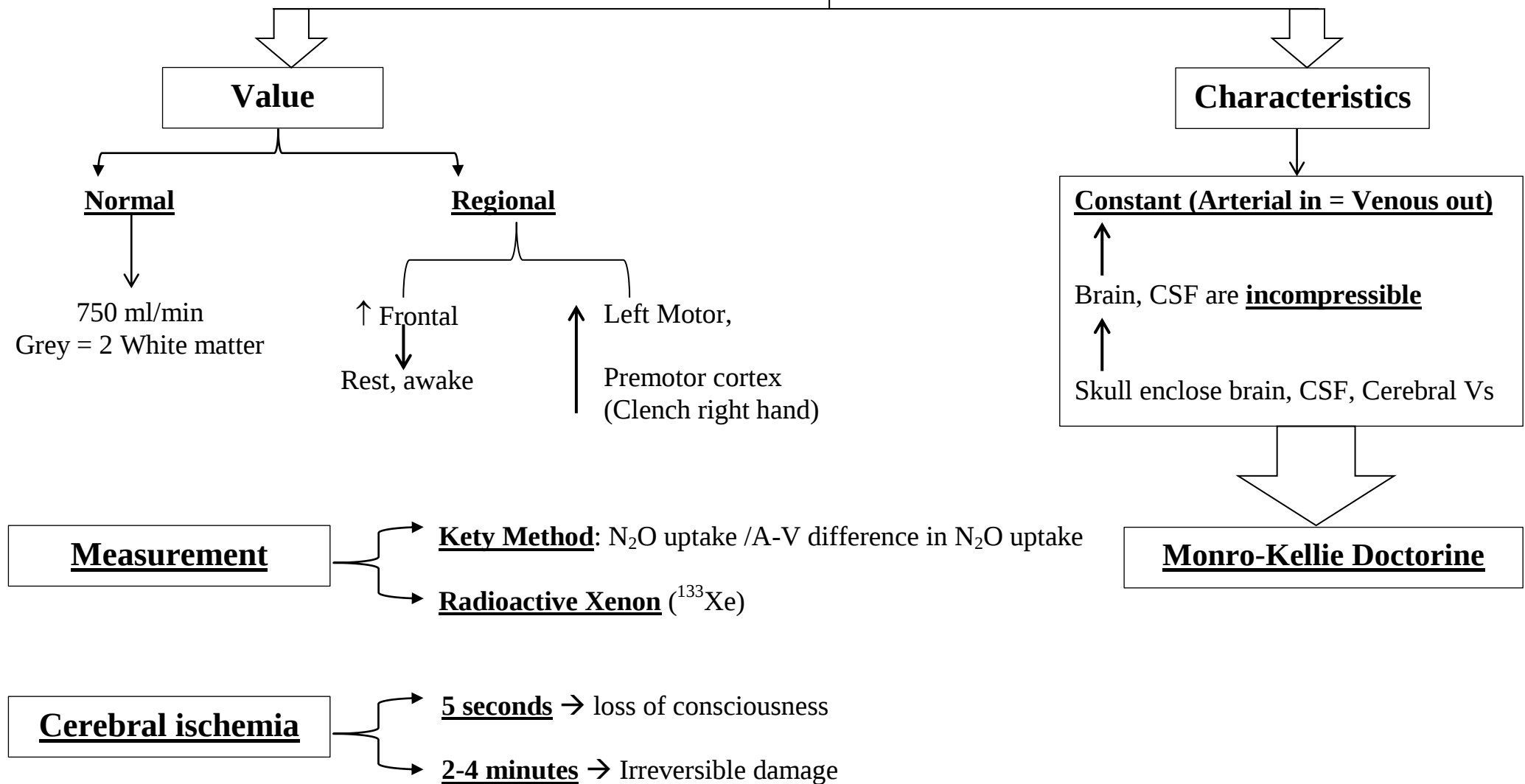
Edema Safety Factors



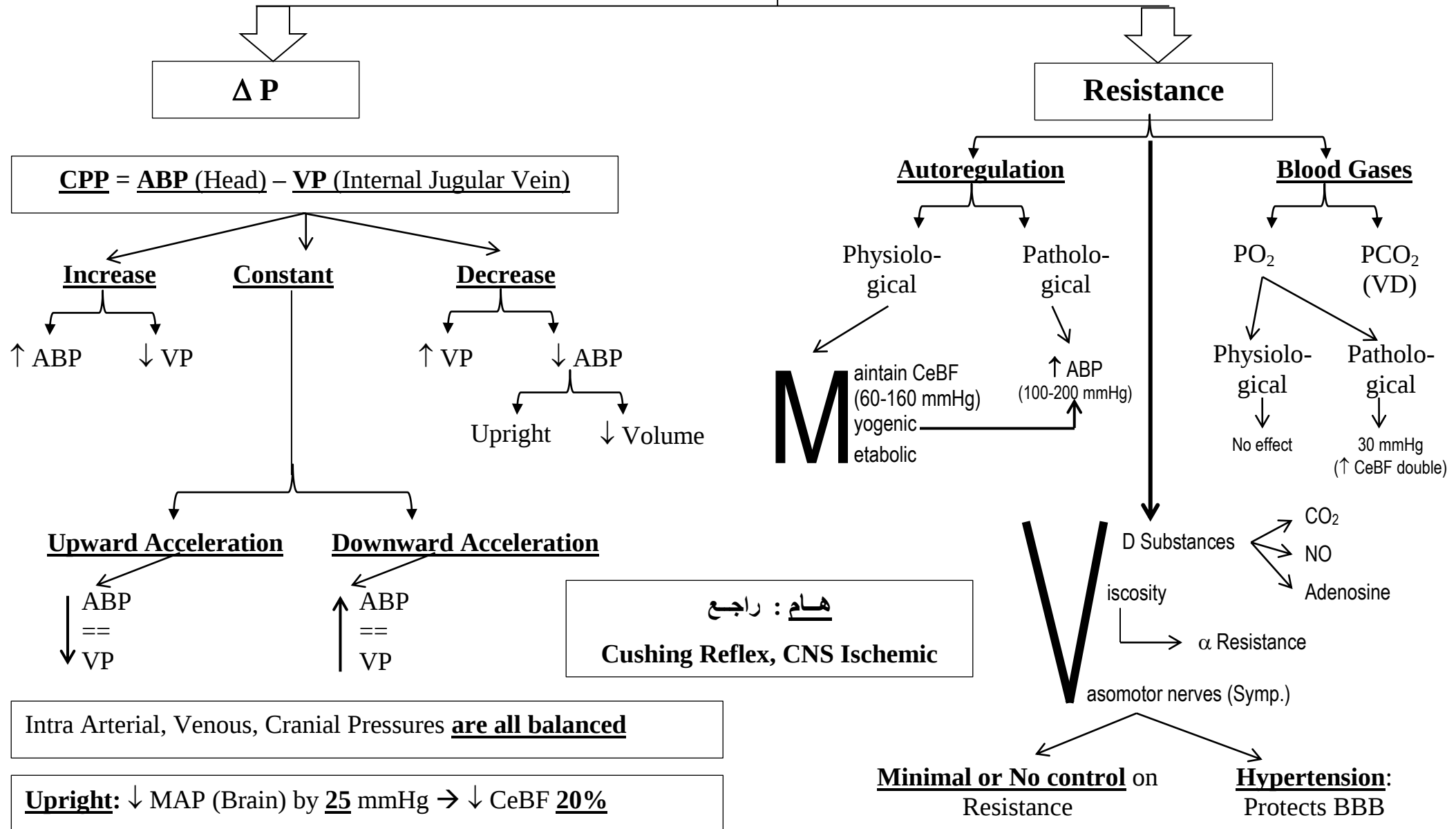
Cerebral Circulation د. محمد فايز



د. محمد فايز Cerebral Blood Flow (CeBF)



د. محمد فايز Factors Affecting Cerebral Blood Flow (CeBF)

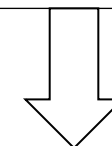


د. محمد فايز Pulmonary Circulation

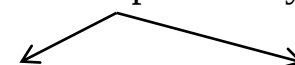


	Pulmonary	Systemic
Flow	More Pulsatile	Less Pulsatile
Pressure:		
SBP	24-25 mmHg	100-140 mmHg
DBP	8-9 mmHg	60-90 mmHg
PP	15 mmHg	30-50 mmHg
PP/SBP	15/25 = 60 %	40/120 = 33%
MAP	15 mmHg	93 mmHg
Hypertension	Mitral stenosis	1 ^{ry} , 2 ^{ry}
MCP	10 mmHg	17 mmHg
Precapillary R.	= Post capillary R	4-5 Post Capil. R
Edema	↑ Capillary P ressure ermeability	
Edema Safety Factors	27 mmHg	17 mmHg
Gravity	<u>Apex</u> : 5 mmHg <u>Mid zone</u> : 15 mmHg <u>Base</u> : 25 mmHg	Each 1 cm below heart level → ↑ Pressure 0.77 mmHg
Resistance	Less Blood Reservoir ↗ 600-700 (Erect) ↘ 1000-1200 (Supine)	High
Respiration	* Inspiration: ↓ VR * Expiration: ↑ VR * ↓ O ₂ → VC of pulmonary artery → ↑ RV press. → RV Hypertrophy (Corpulmonale)	* Inspiration: ↑ VR * Expiration: ↓ VR * ↓ O ₂ → VD

Transient Time

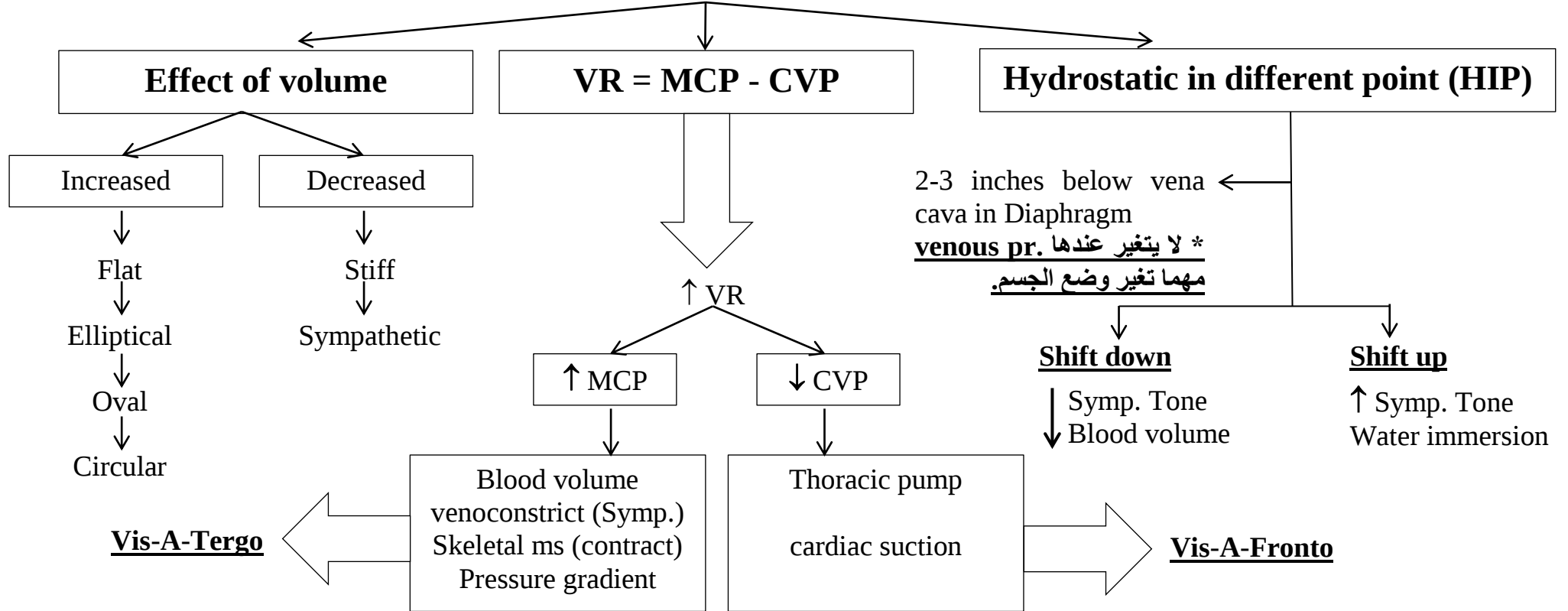


Time which **RBC take** to
Traverse pulmonary Cap.



0.75 sec. (Rest)
0.3 sec. (Exerc.)

د. محمد فايز Venous Circulation



Venous Pressure

- 0-2 (vena cava)
- 6-8 (large veins)
- 8-10 (small veins)
- 10-12 (venules)

Rapid venous velocity (↓ CSA)

Reference Point

4th sternocostal joint (middle of Rt atrium = **CVP = Zero**).

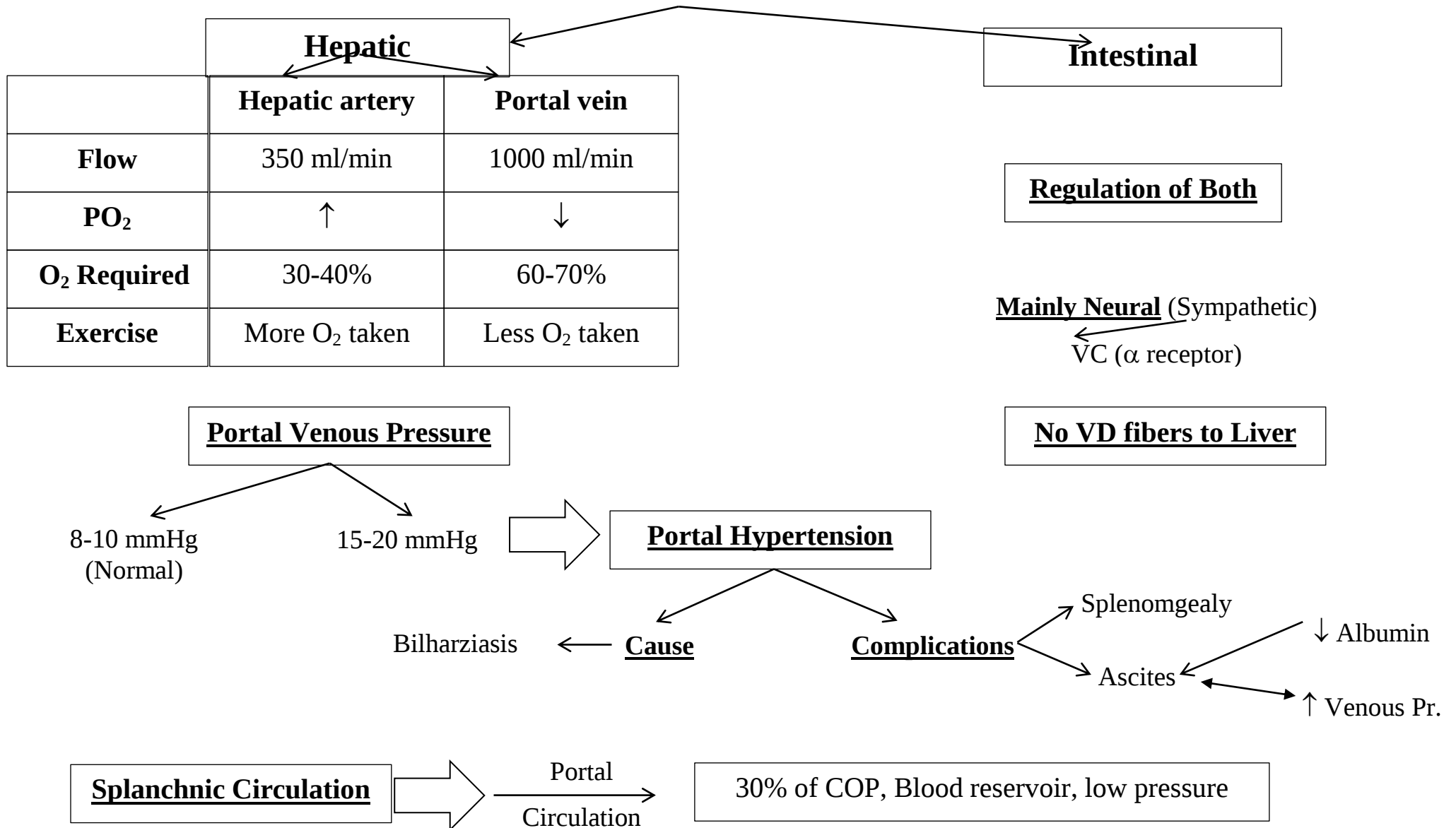
Flow of veins (2 Descent)



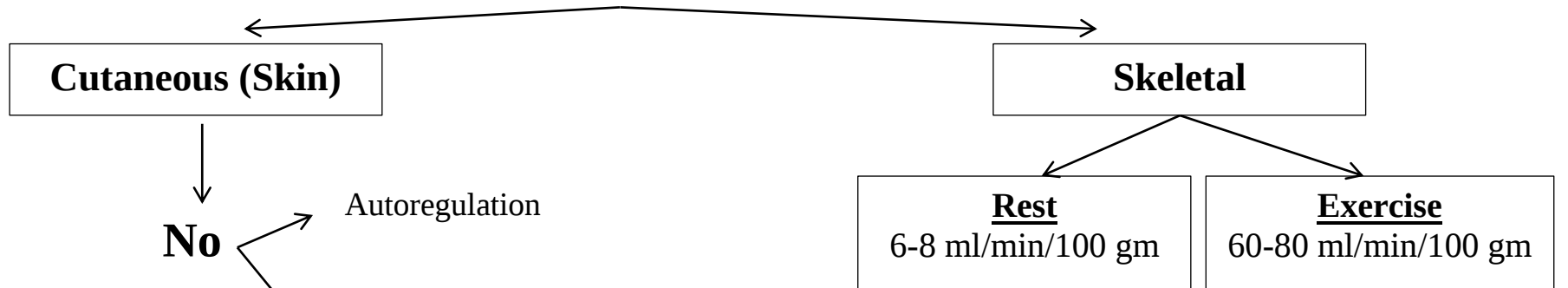
Cardiac activity α VR



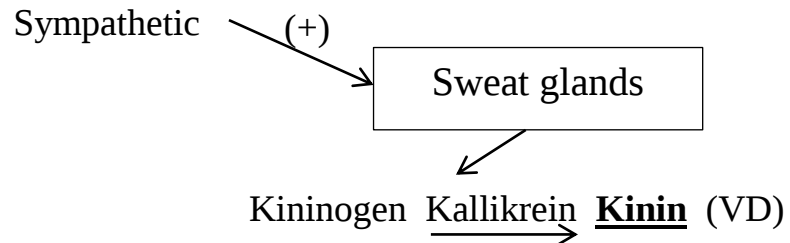
د. محمد فايز Splanchnic Circulation



د. محمد فايز Other Circulations

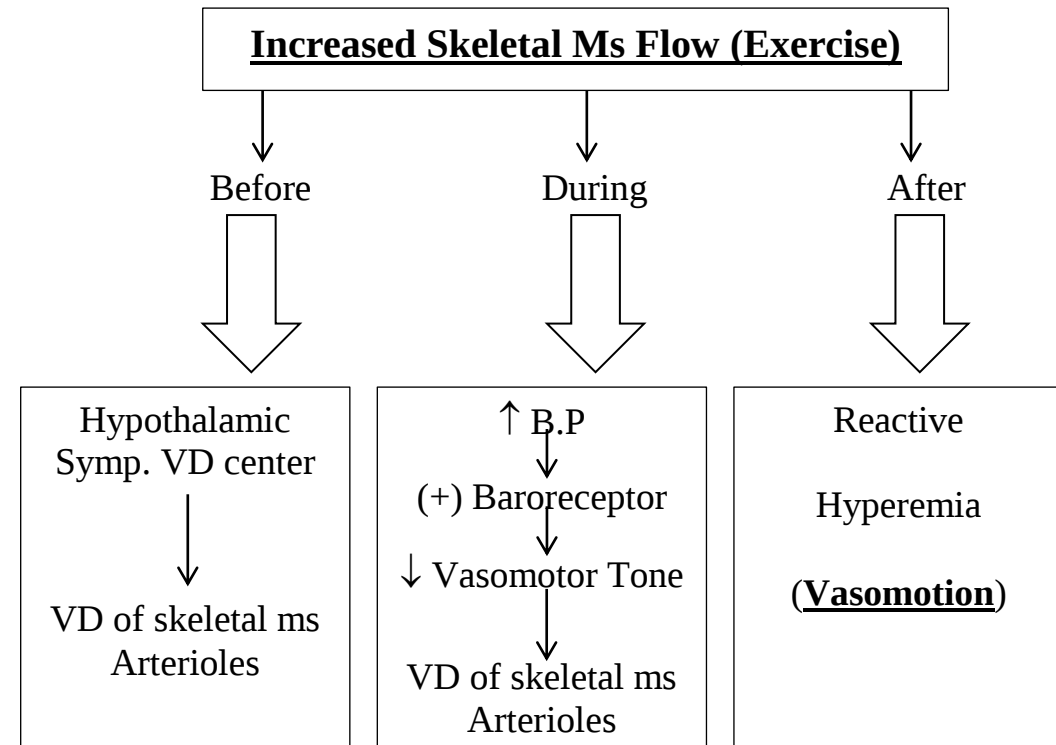


سؤال: إزاي يحصل VD ؟



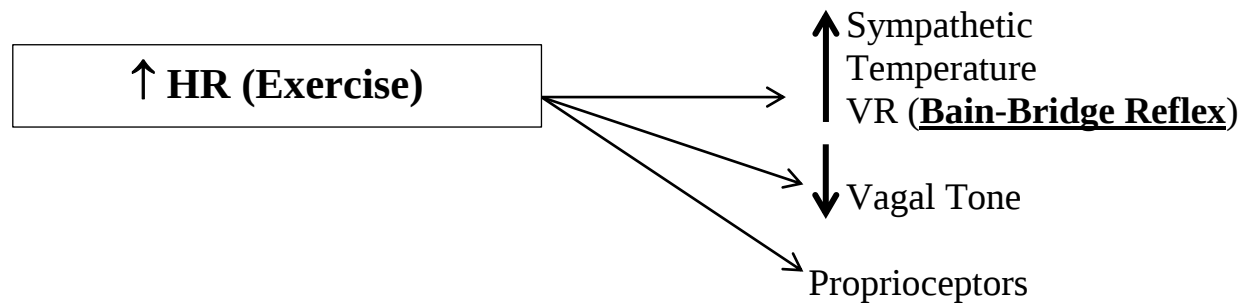
Triple Response

	Red line	Flare	Wheal
Duration	5-10 seconds	30-45 seconds	3 minutes
Cause	Capillary dilate	Local Axon R.	↑ Capillary Permeability



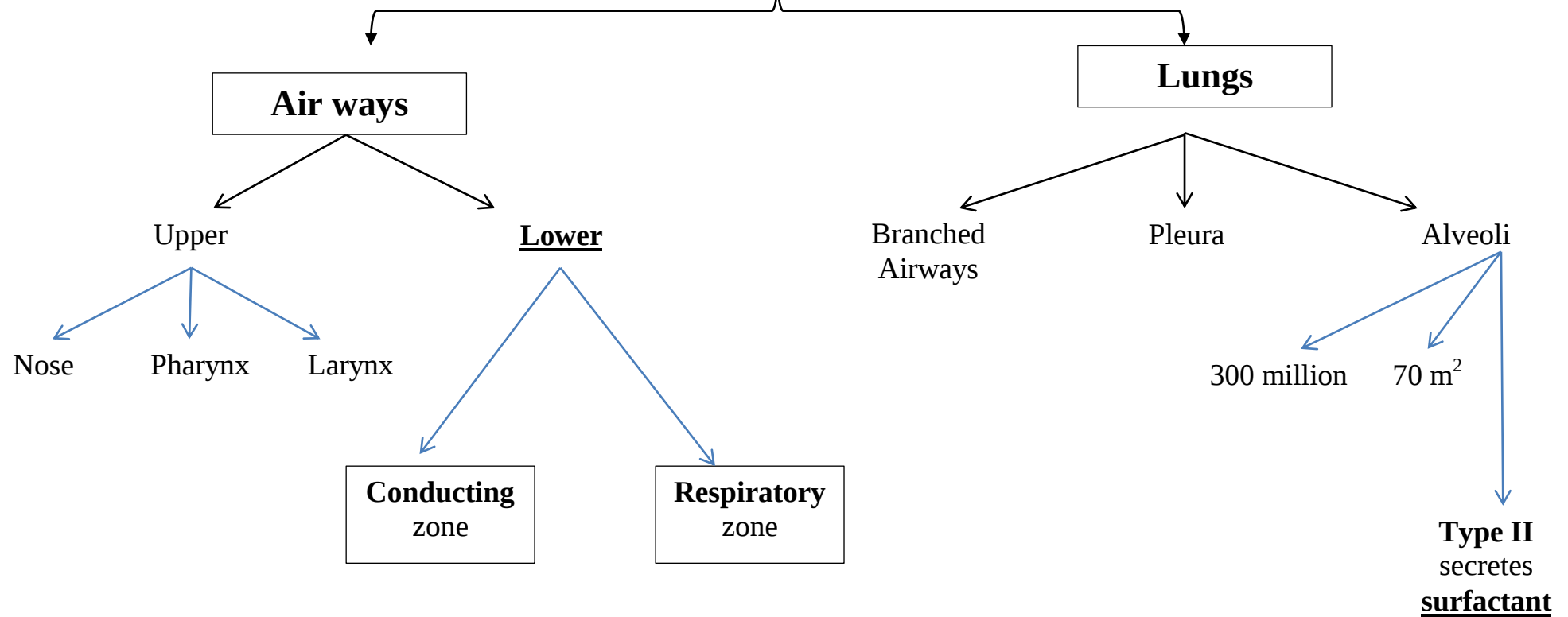
د. محمد فايز Circulatory Response to Exercise

	Isometric	Isotonic
Blood flow	↓	↑
HR	↑	
SV	Little change	
SBP	↑	
DBP		↓
PP	No or little change	↑
MAP	↑	The same



Respiration

Respiratory System د. محمد فايز



Site Trachea → Terminal

Resp. bronchioles → Alveolar sac

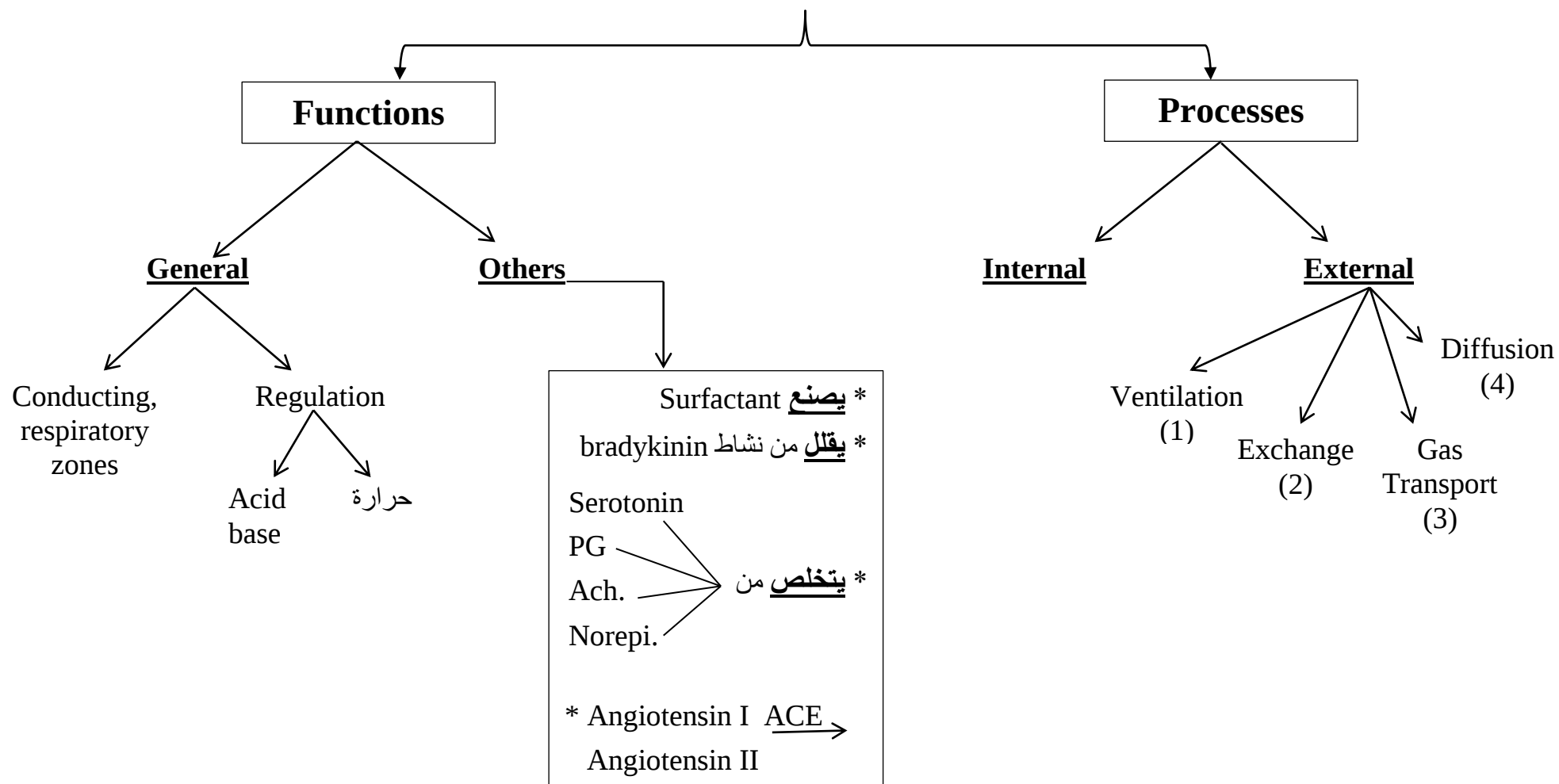
Alveoli --

+

Functions Air (conduct, warm, moisten), Defencive + Phonation

Gas exchange

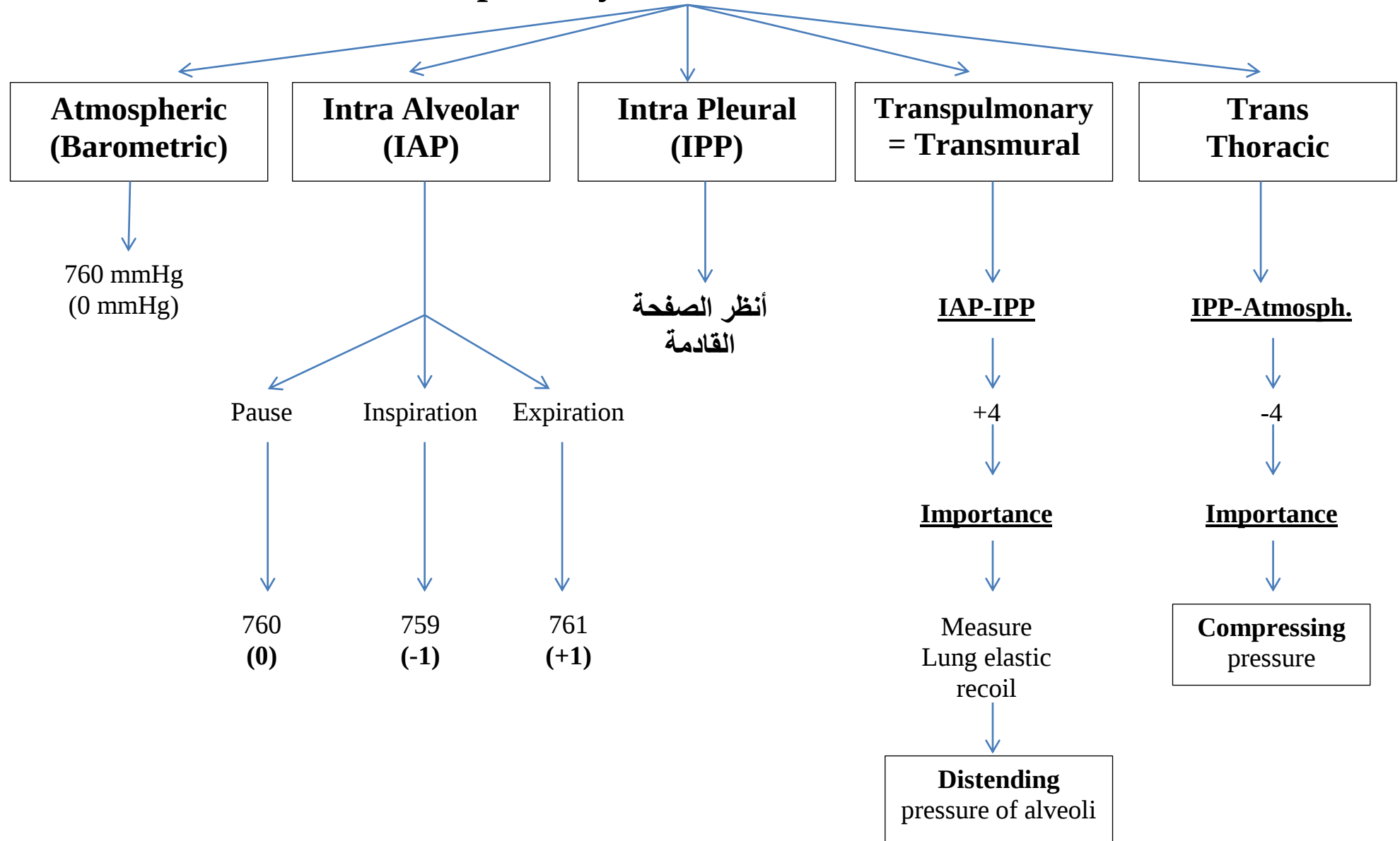
د. محمد فايز Respiratory



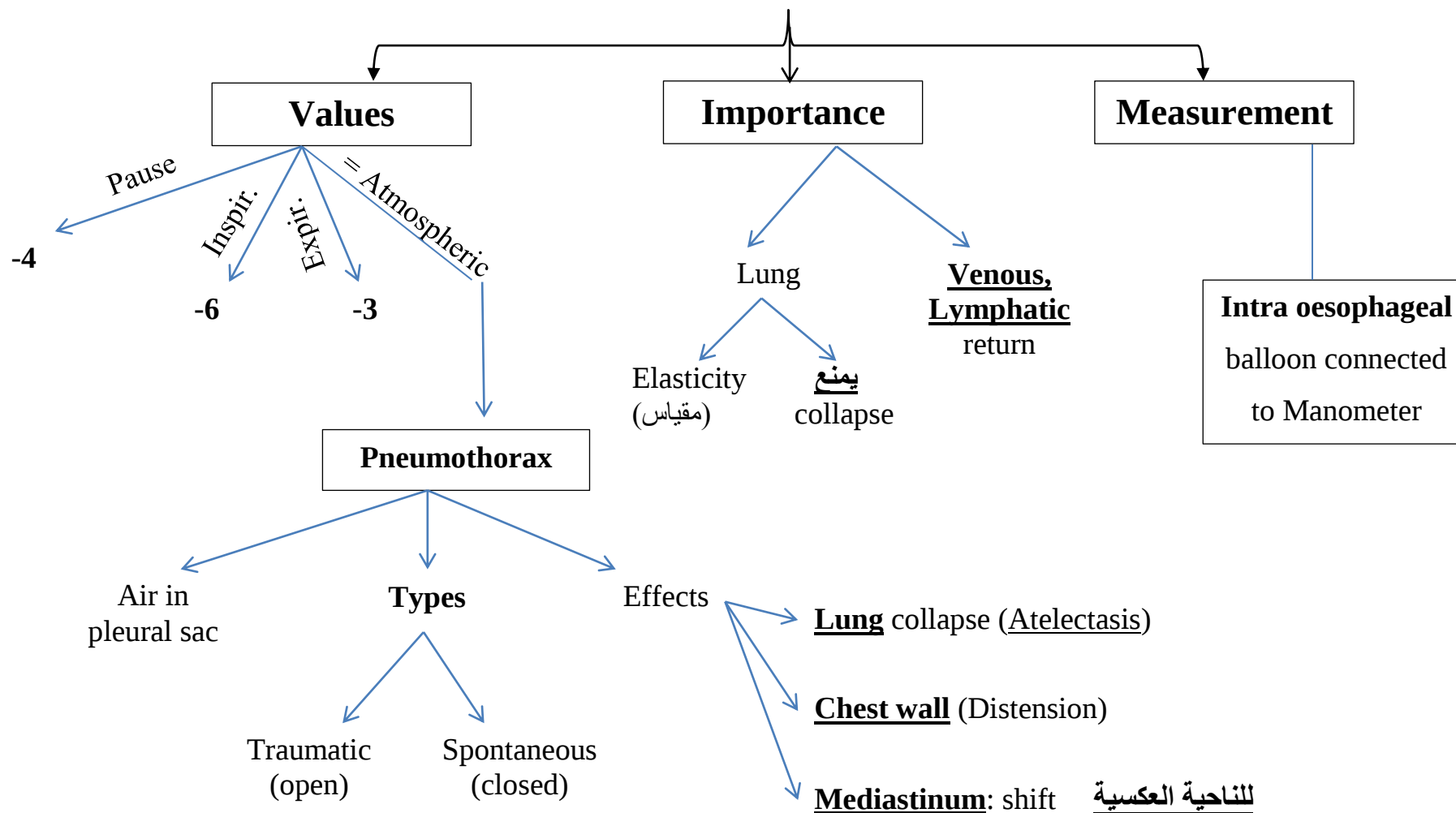
د. محمد فايز Mechanism of Respiration

	Inspiration	Expiration
Lung	<u>Start</u> : Tend to recoil <u>End</u> : Distension	<u>Start</u> : Distension <u>End</u> : Recoil
Thoracic cavity	↑ all Dimensions	↓ all Dimensions
IPP	-6	-3
IAP	-1	+1
Air moves	in	out
Respiratory Muscles		
Normal	1. Diaphragm (75%) ↑ Vertical 2. External Intercostal: ↑ Transverse, AP Diameter	Relaxation of inspiratory muscles
Deep (Forced)	Accessory Muscles ↓ Sternomastoid calenus	1. Internal Intercostal (External عكس) 2. Abdominal Muscles ↓ Vertical diameter

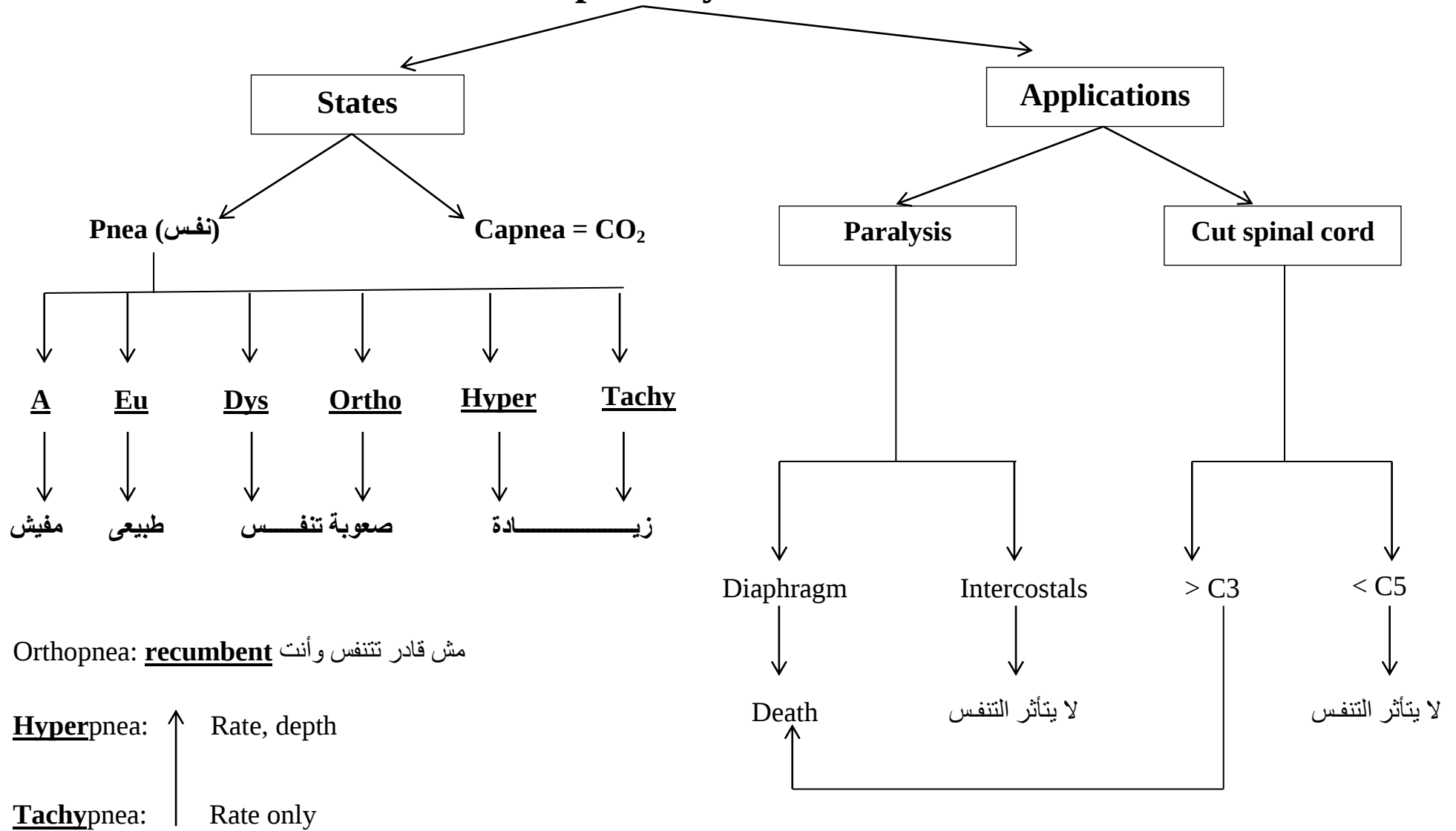
Respiratory Pressures د. محمد فايز



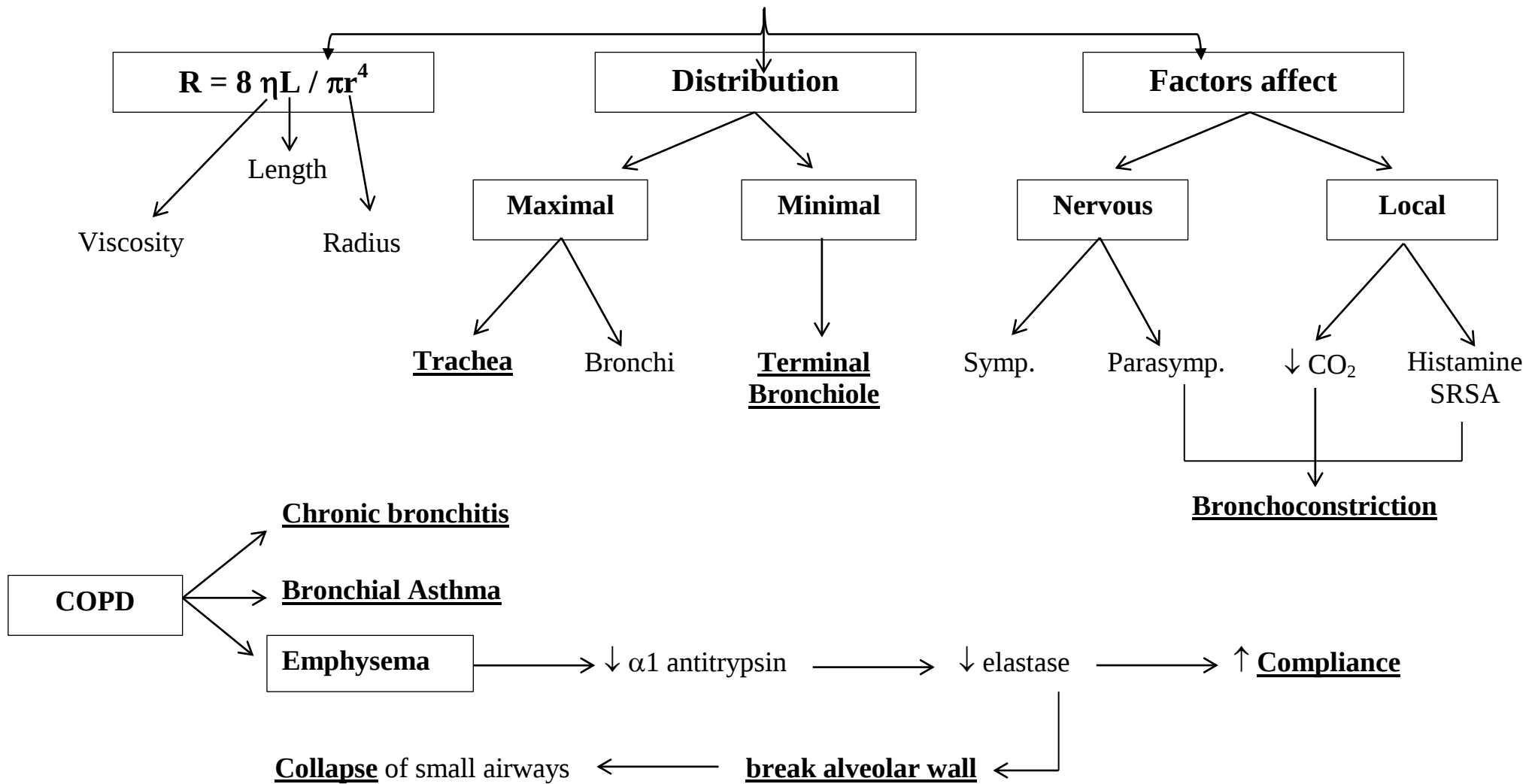
د. محمد فايز Intra-pleural Pressure (IPP)



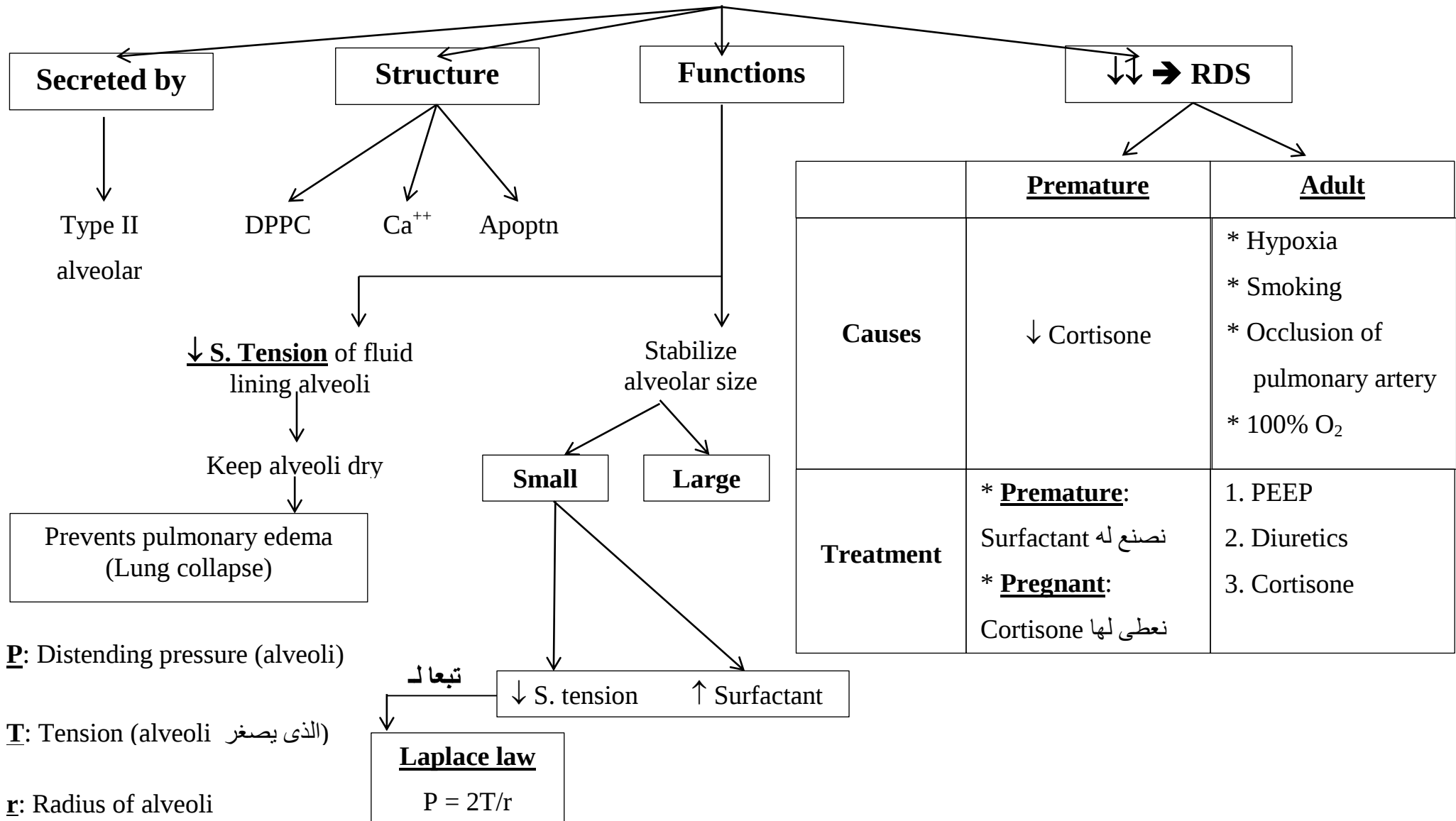
د. محمد فايز Respiratory



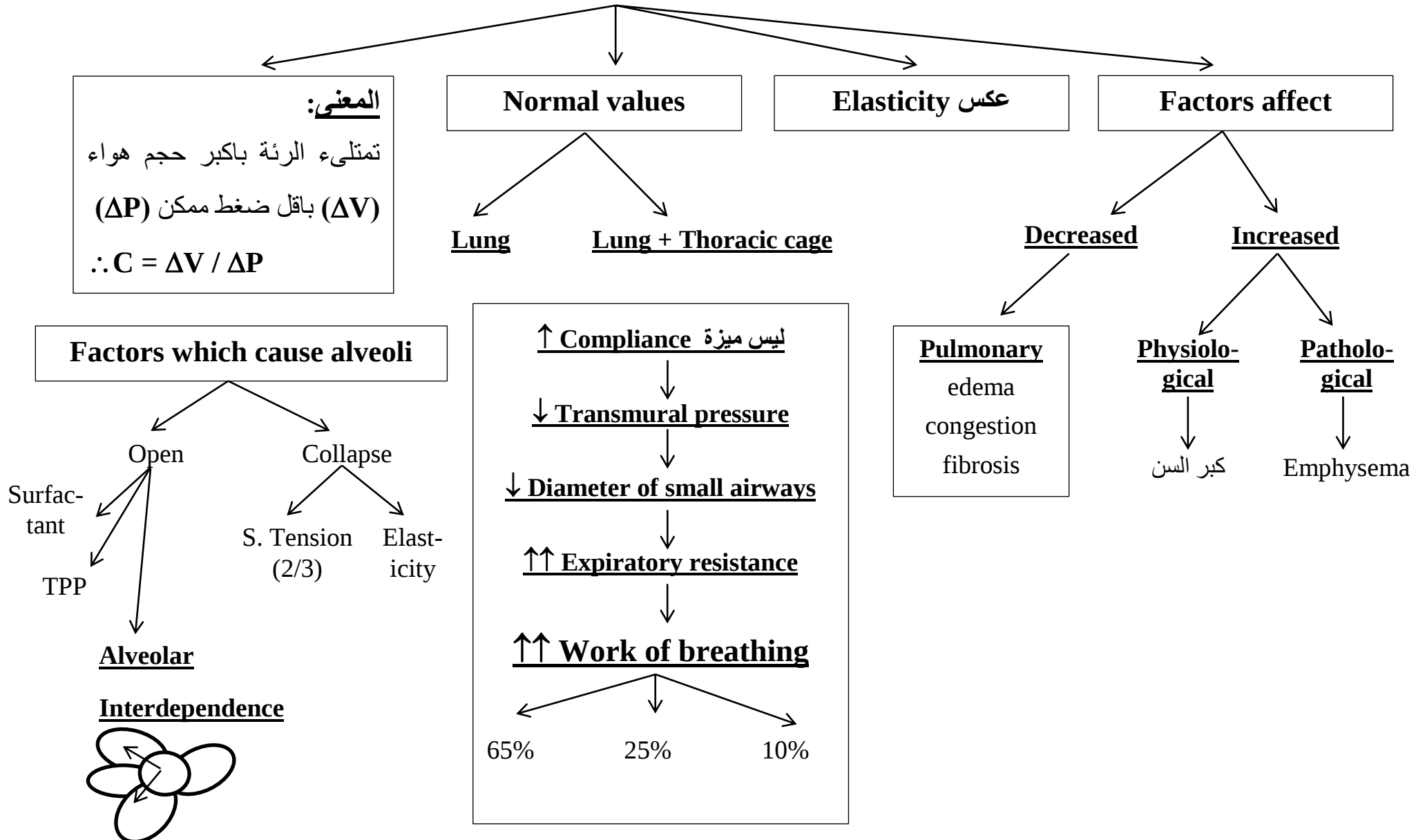
Airway Resistance د. محمد فايز



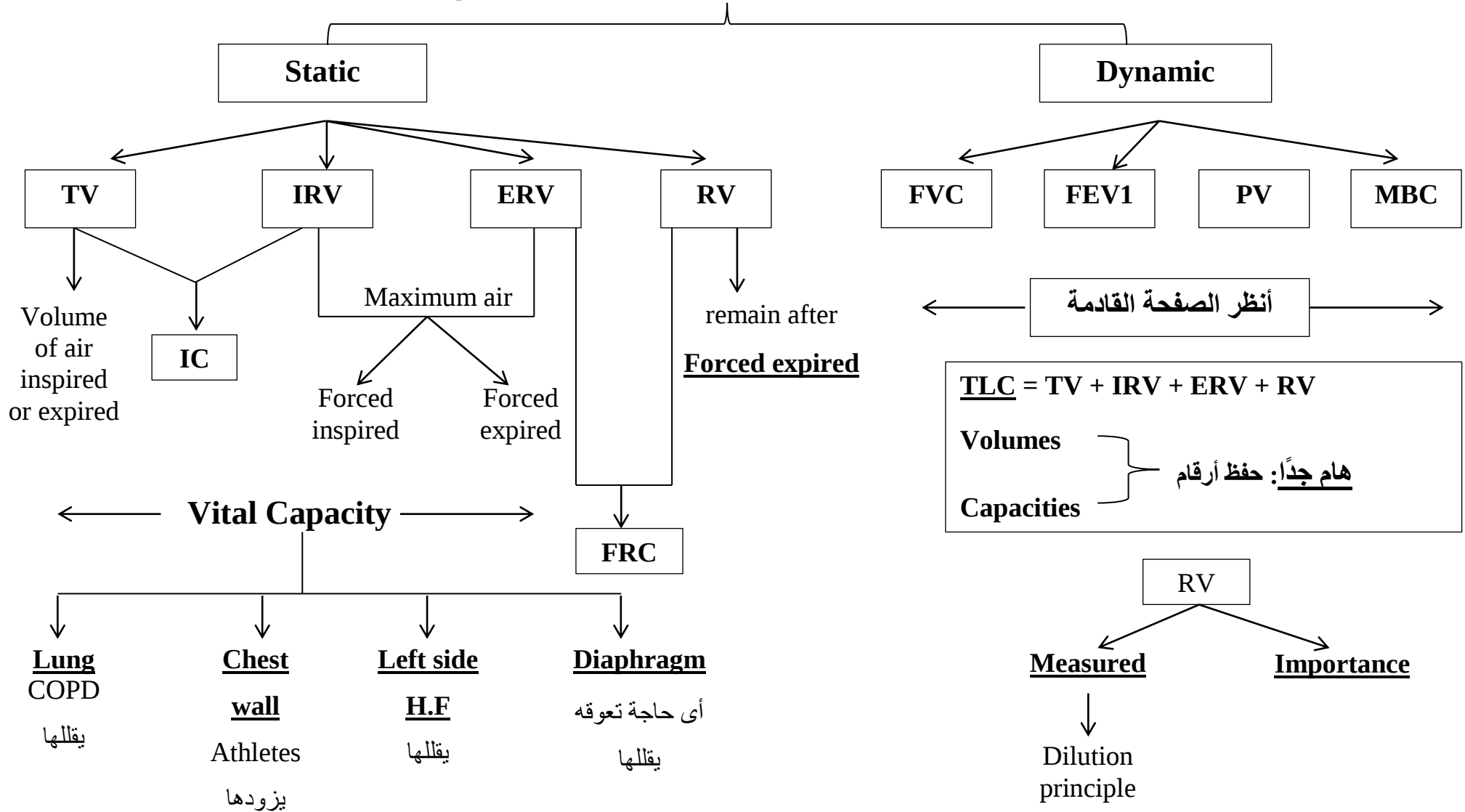
د. محمد فايز Surfactant



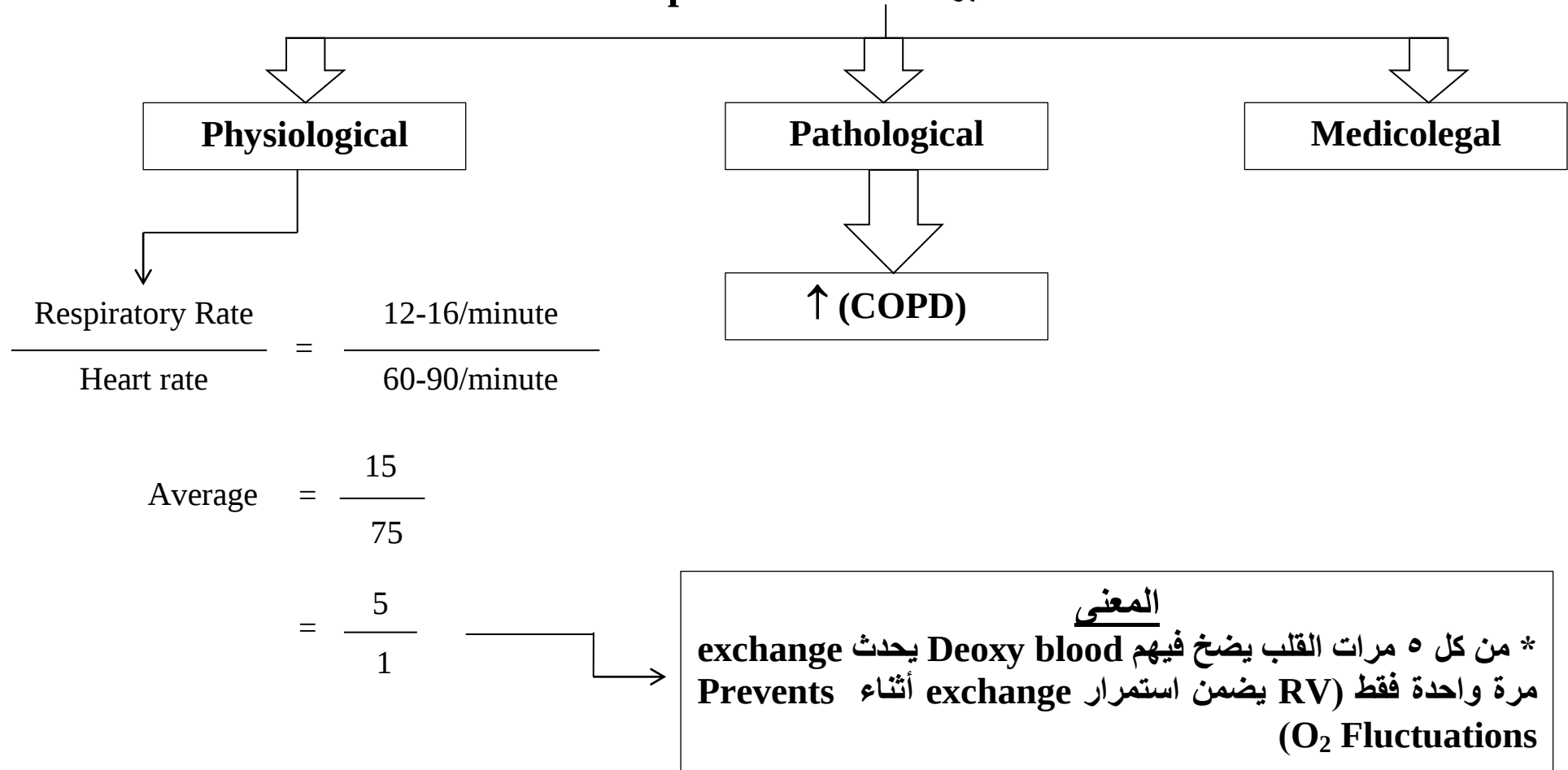
د. محمد فايز Compliance (C)



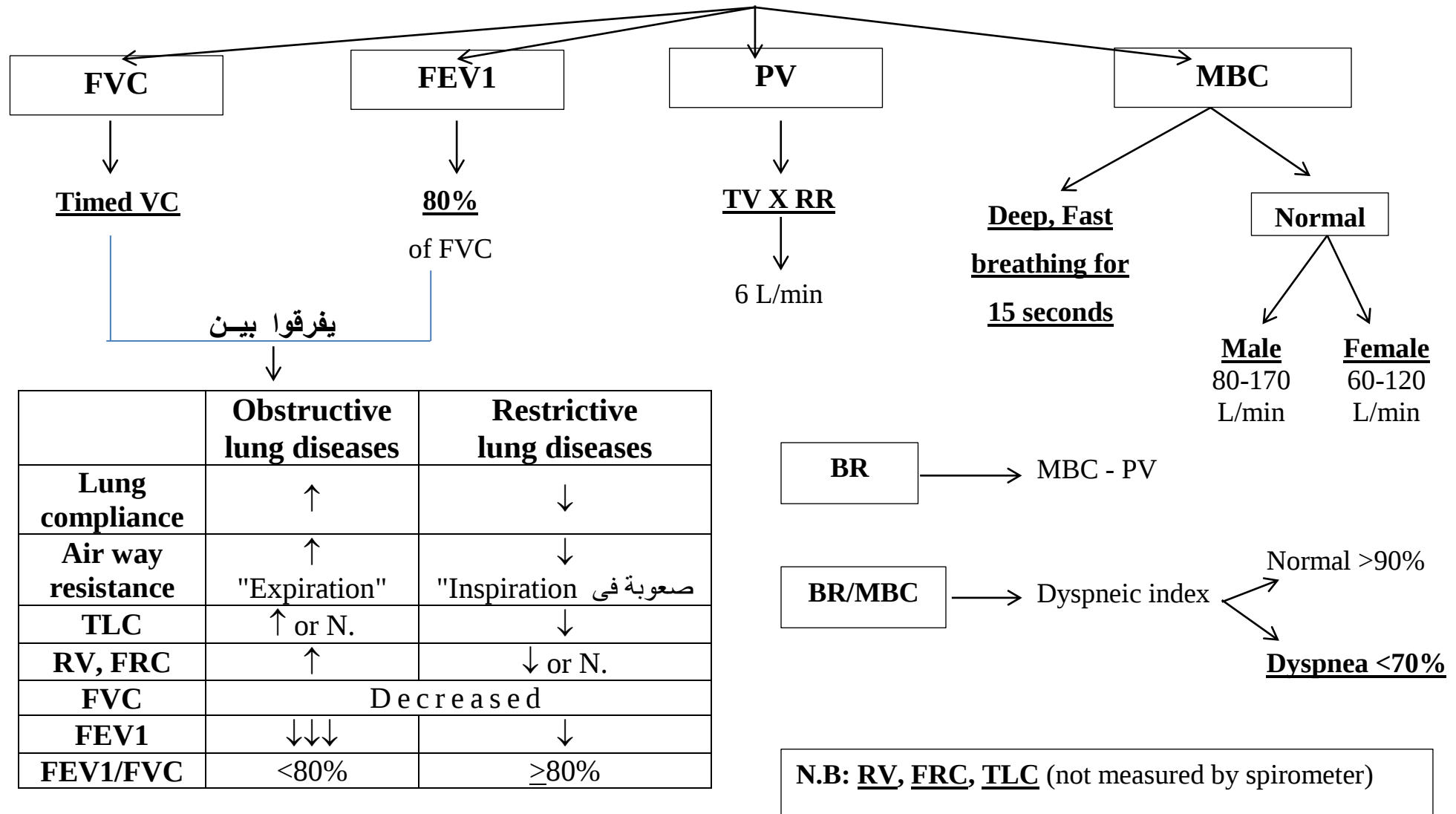
د. محمد فايز Lung Volumes and Capacities



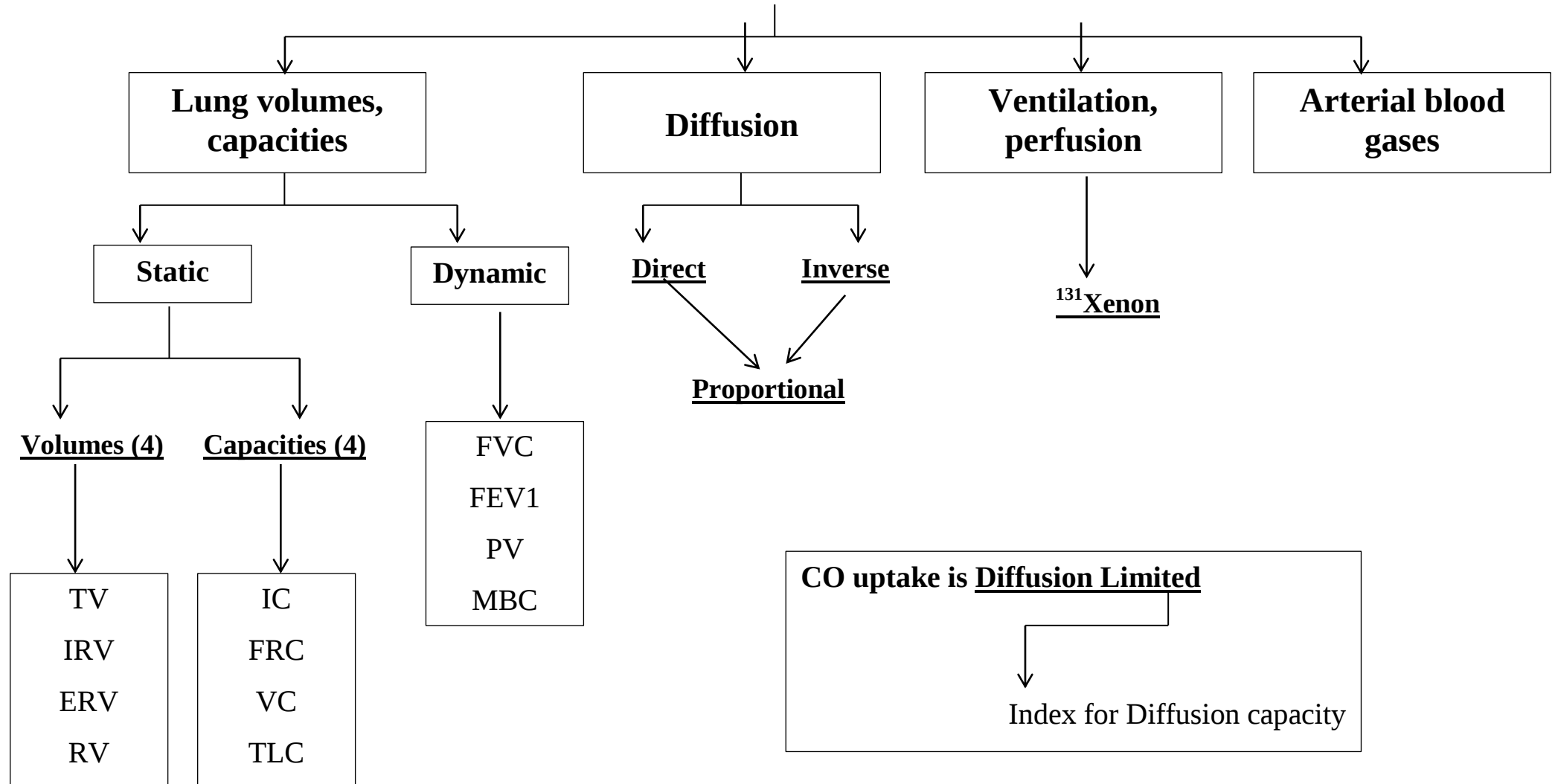
د. محمد فايز Importance of RV



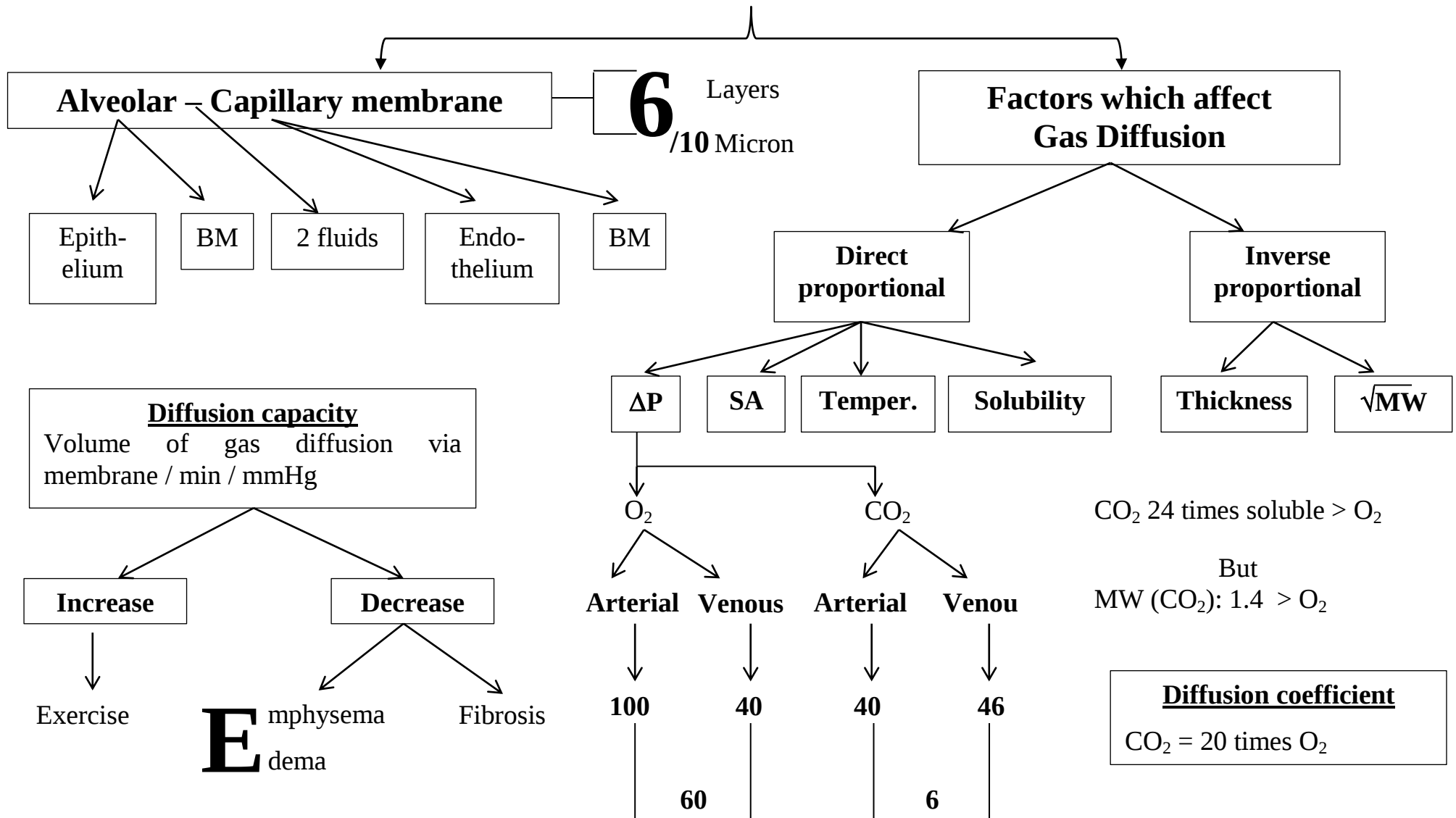
د. محمد فايز Dynamic Lung Volumes



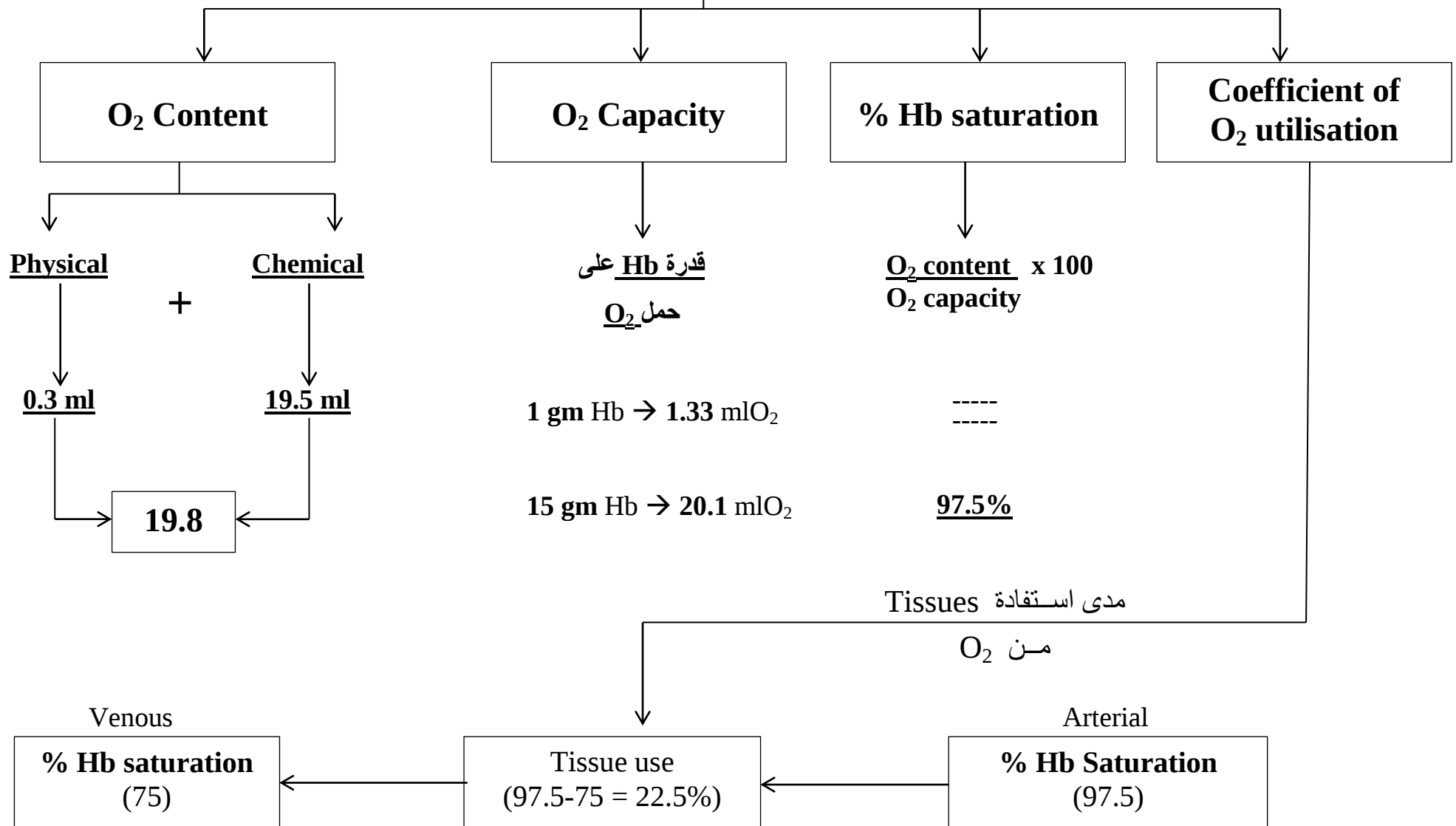
Pulmonary Function Tests د. محمد فايز



Gas Exchange د. محمد فايز



O₂ Transport د. محمد فايز



د. محمد فايز O₂ Dissociation Curve

أنظر الصفحة القادمة

HB

Myoglobin

Plateau part of curve

PO₂ (100 → 60)

% Hb saturation (97.5 → 90)

Significance

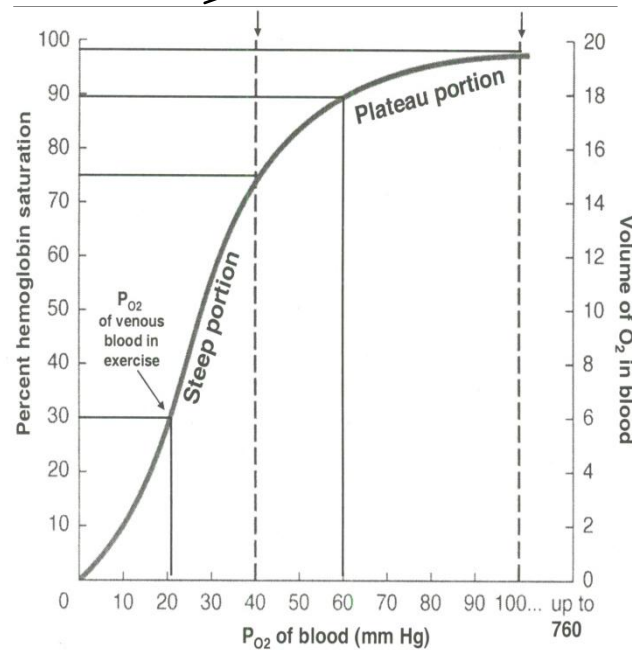
Physiological

Pathological

High altitude

Lung diseases

Safety against PO₂ changes



Steep part of curve

PO₂

% Hb

< 60 → ↓ rapidly

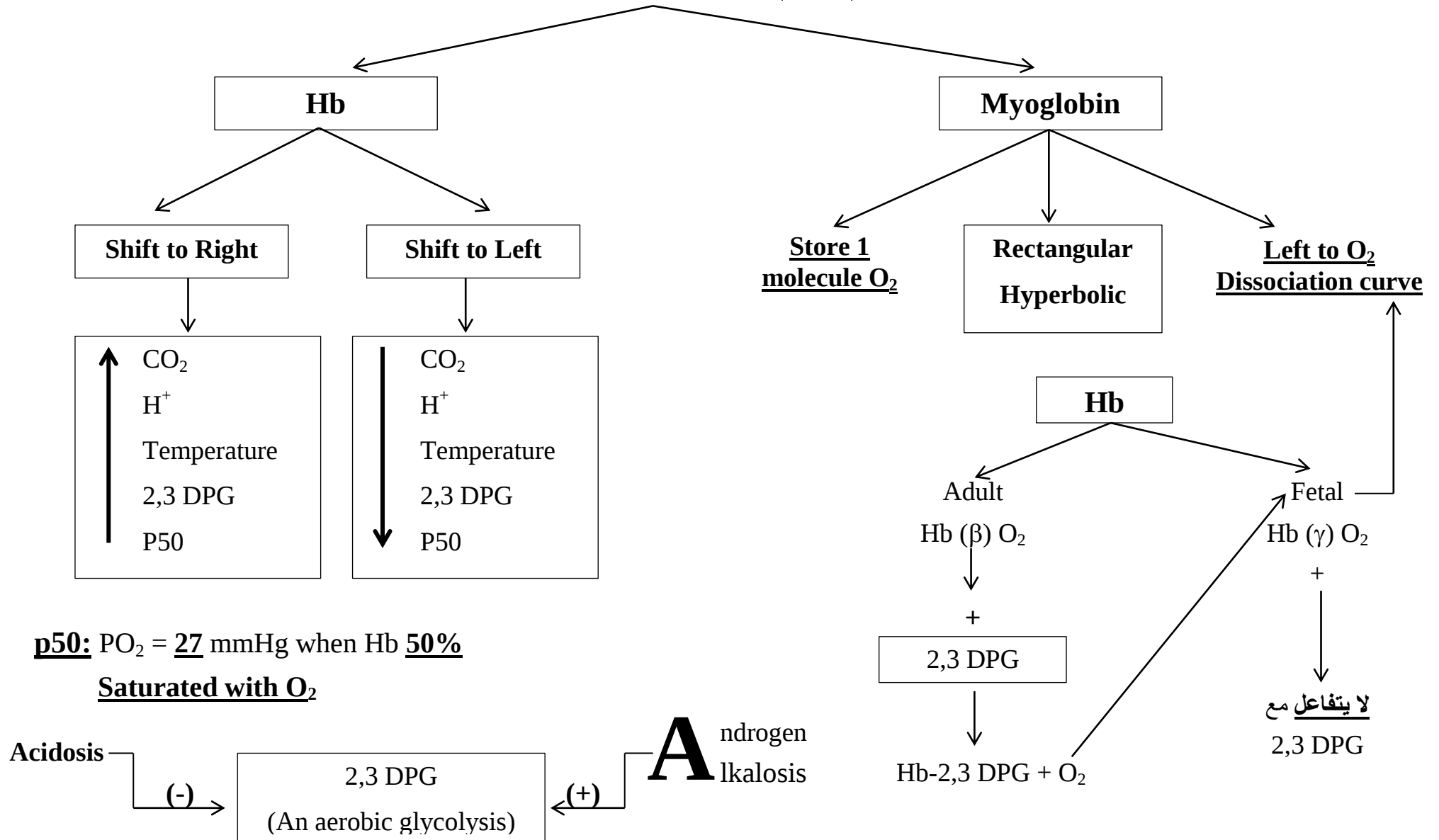
40 → 75

20 → 30

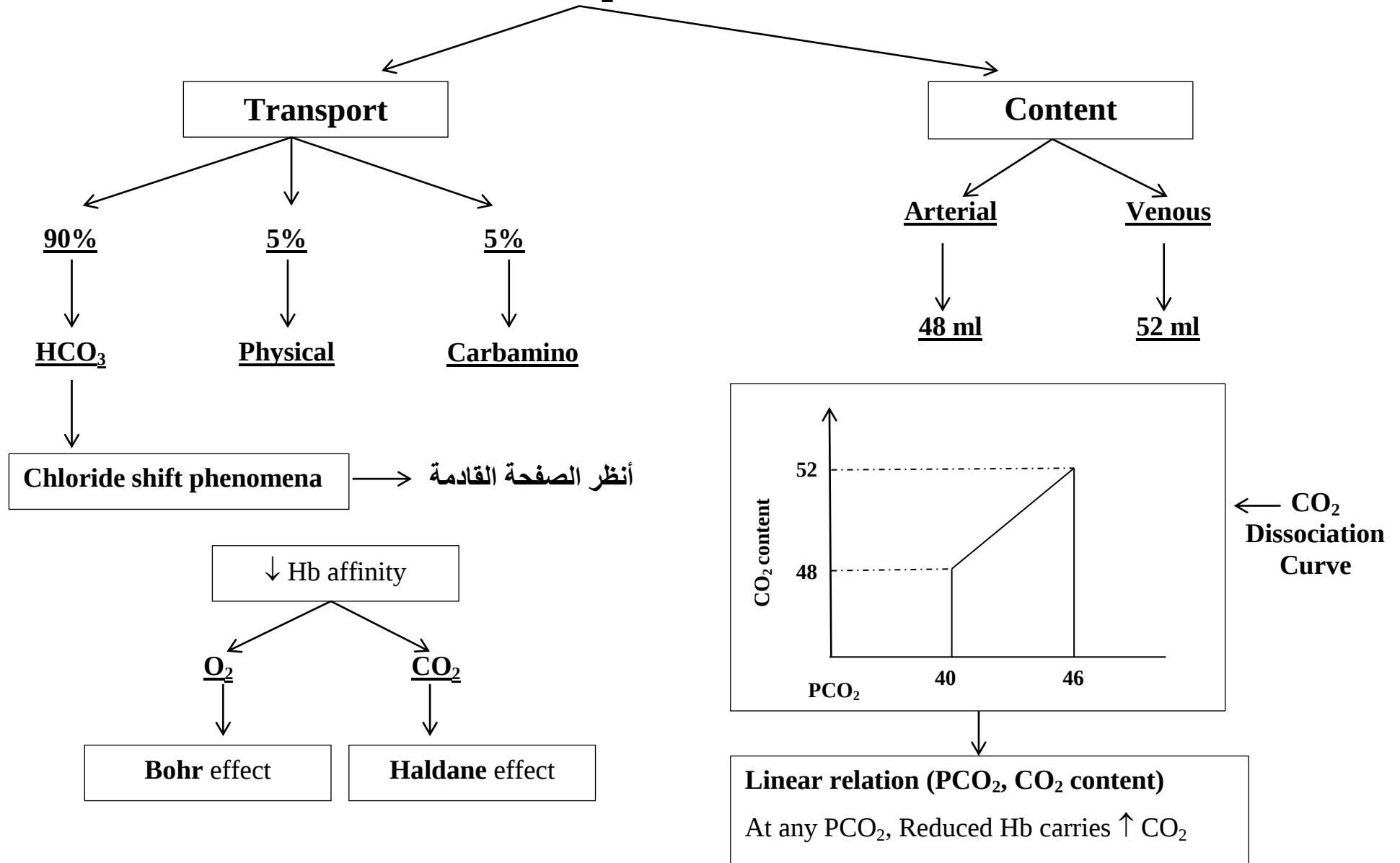
Significance

Small ↓ PO₂ → ↑↑ O₂ release at tissues

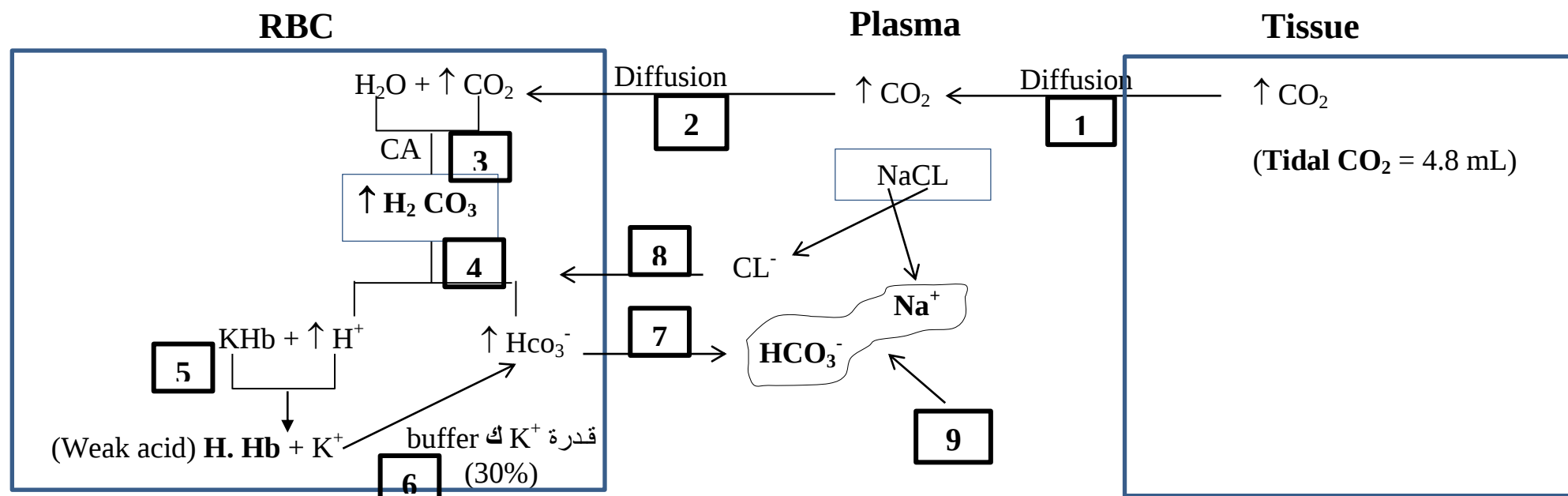
O₂ Dissociation Curve (تكملة) د. محمد فايز



د. محمد فايز CO_2



Chloride shift phenomena at Tissue د. محمد فايز



Note

→ Number of ions (RBCs) > Plasma → ↑ Osmotic pressure → attract water to move inside RBC → ↑ HV (**Venous > Arterial blood**)

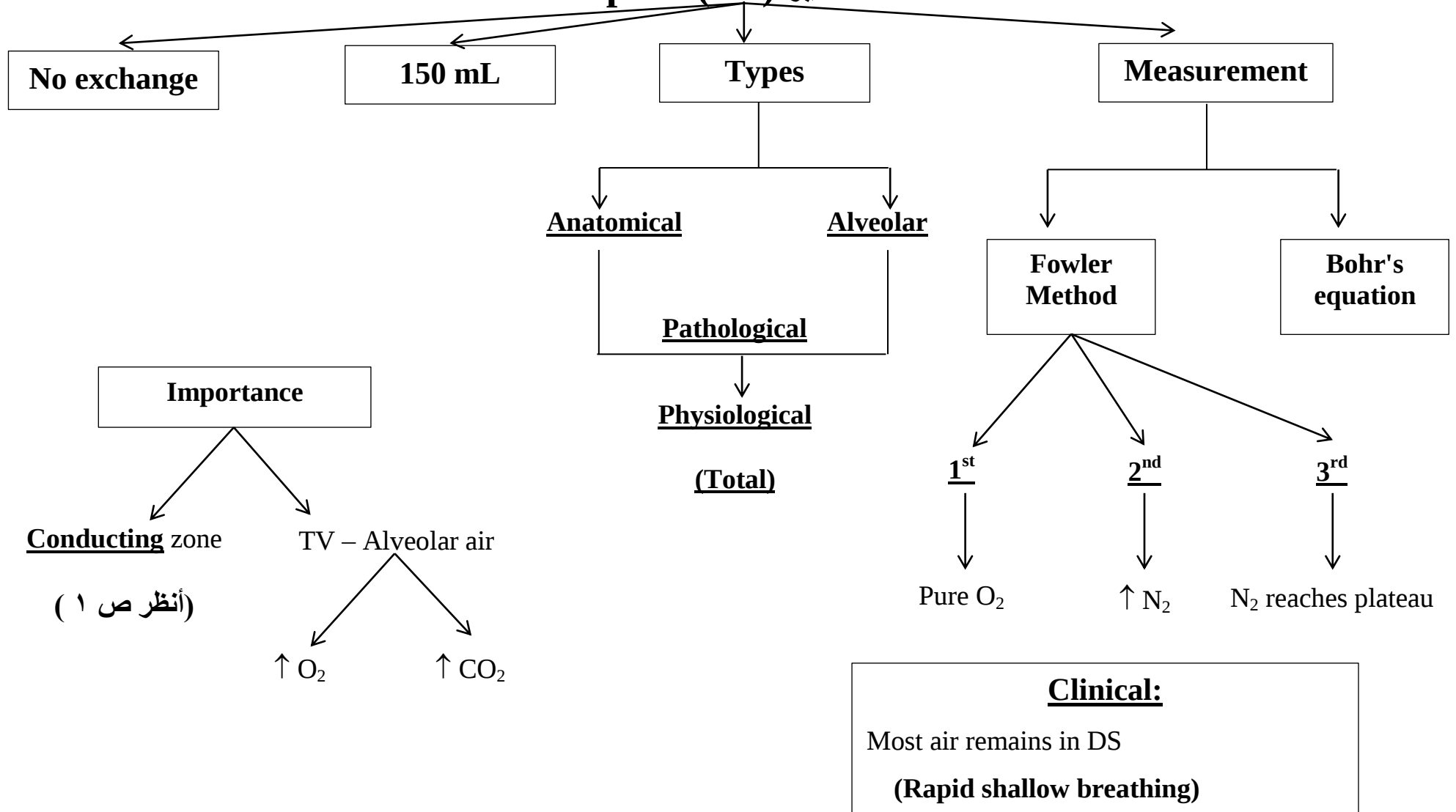
سؤال هام على

Chloride shift phenomena at lung
is the reverse

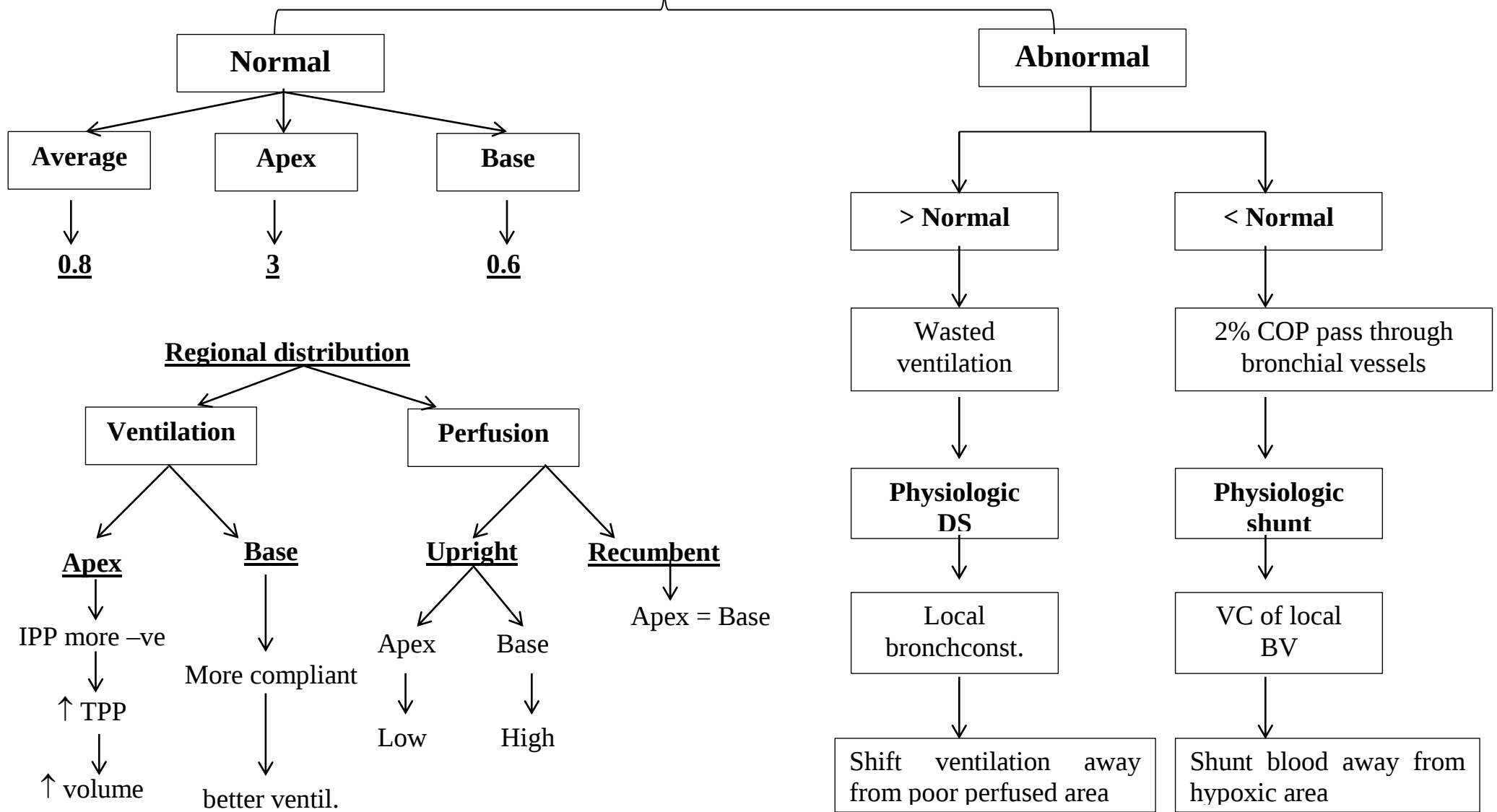
هام

الارقام تعبر عن ترتيب الأحداث

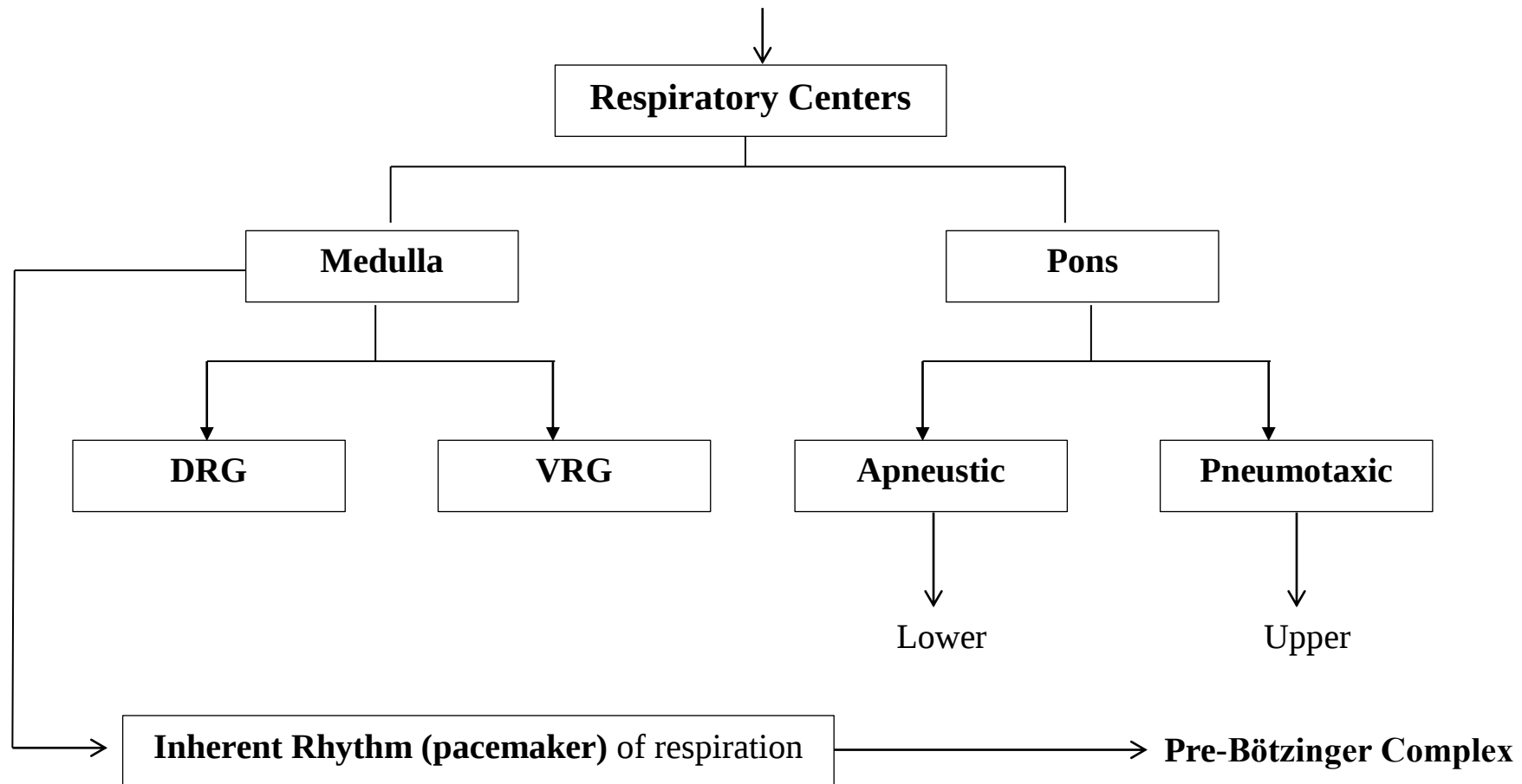
Dead Space (DS) د. محمد فايز



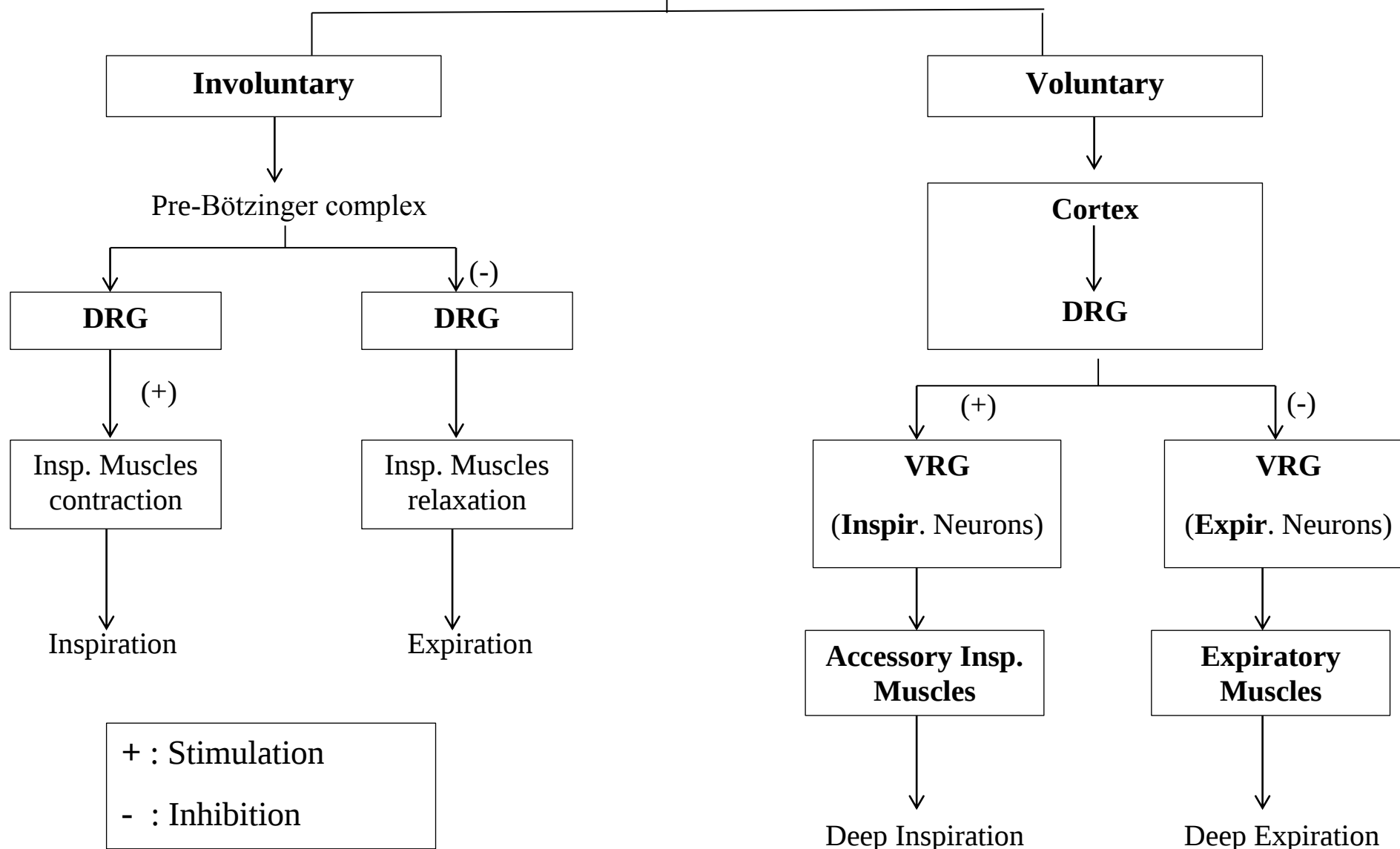
د. محمد فايز Ventilation/Perfusion Ratio



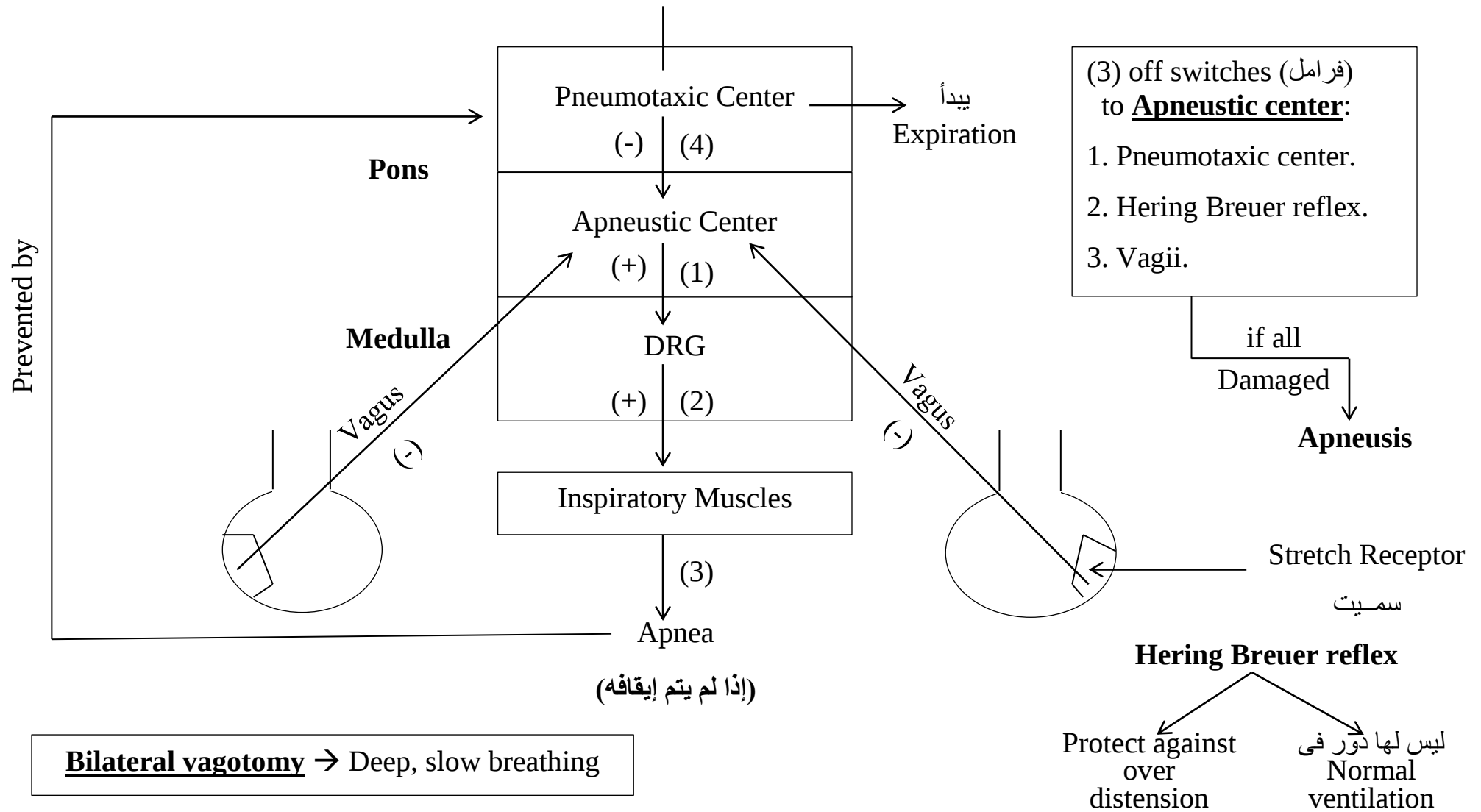
د. محمد فايز Control of Respiration



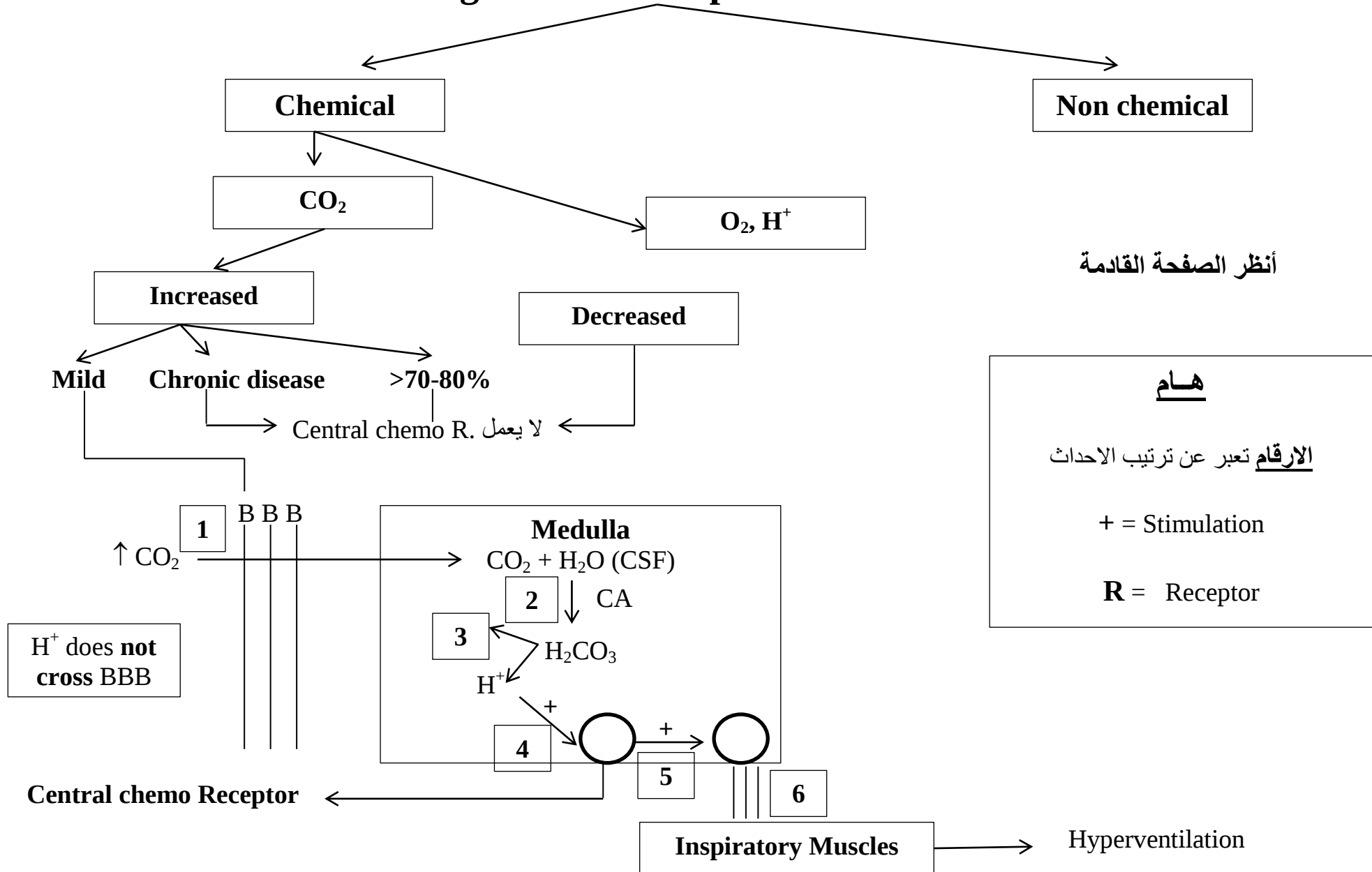
د. محمد فايز Control of Respiration



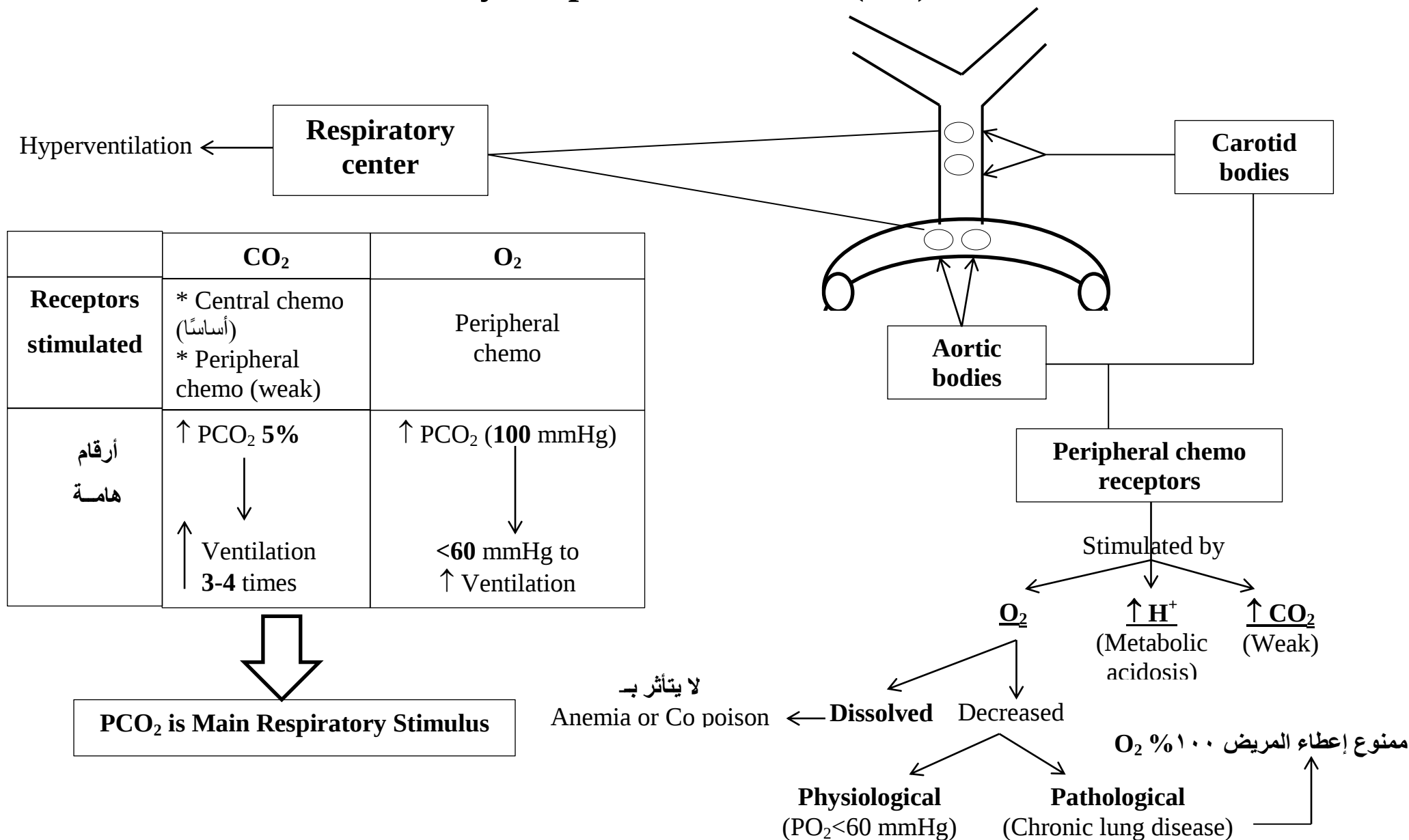
د. محمد فايز Pontine Centers



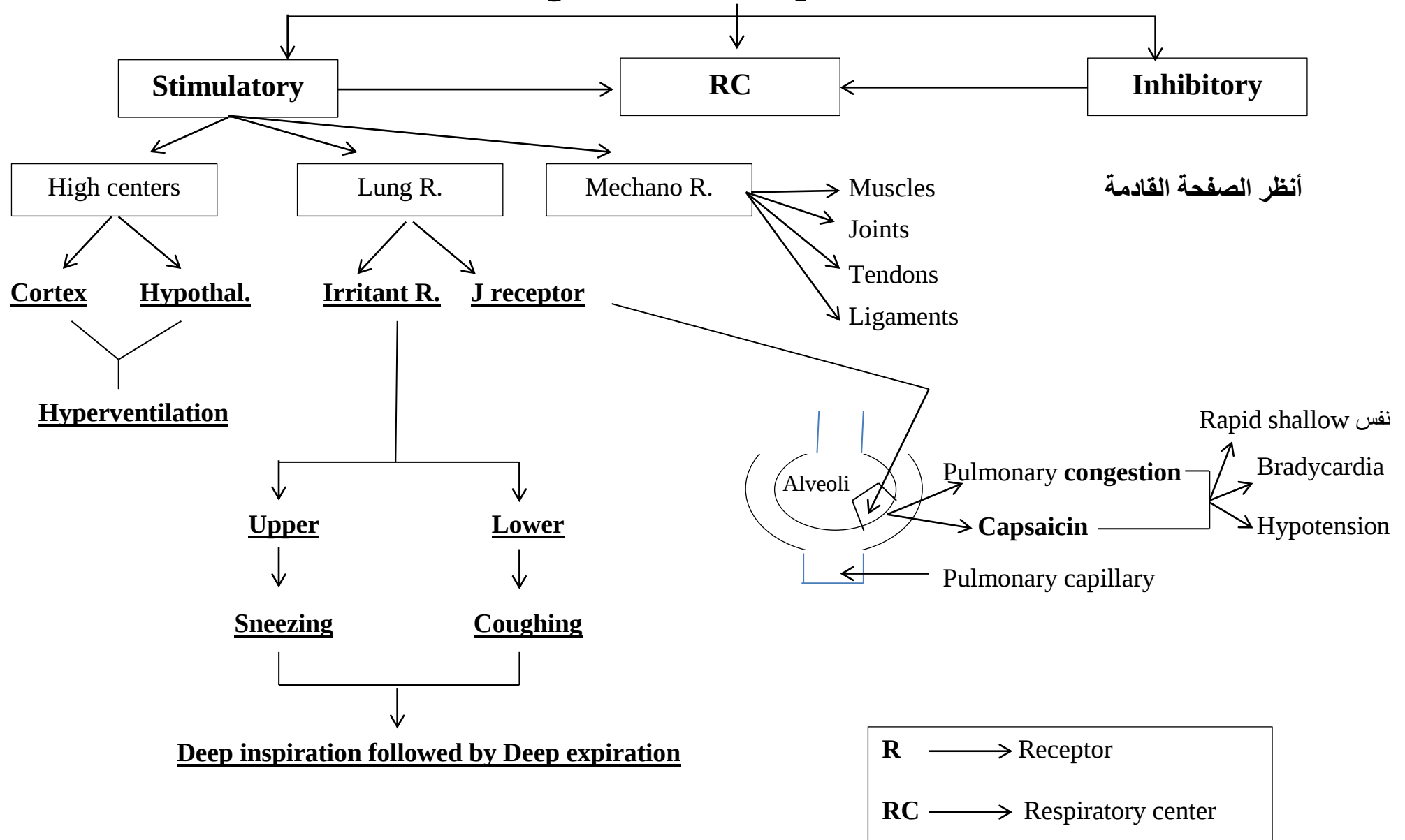
Regulation of Respiration د. محمد فايز



د. محمد فايز Ventilatory Response to PO_2 , H^+ (PH)



Non Chemical Regulation of Respiration د. محمد فايز



أنظر الصفحة القادمة

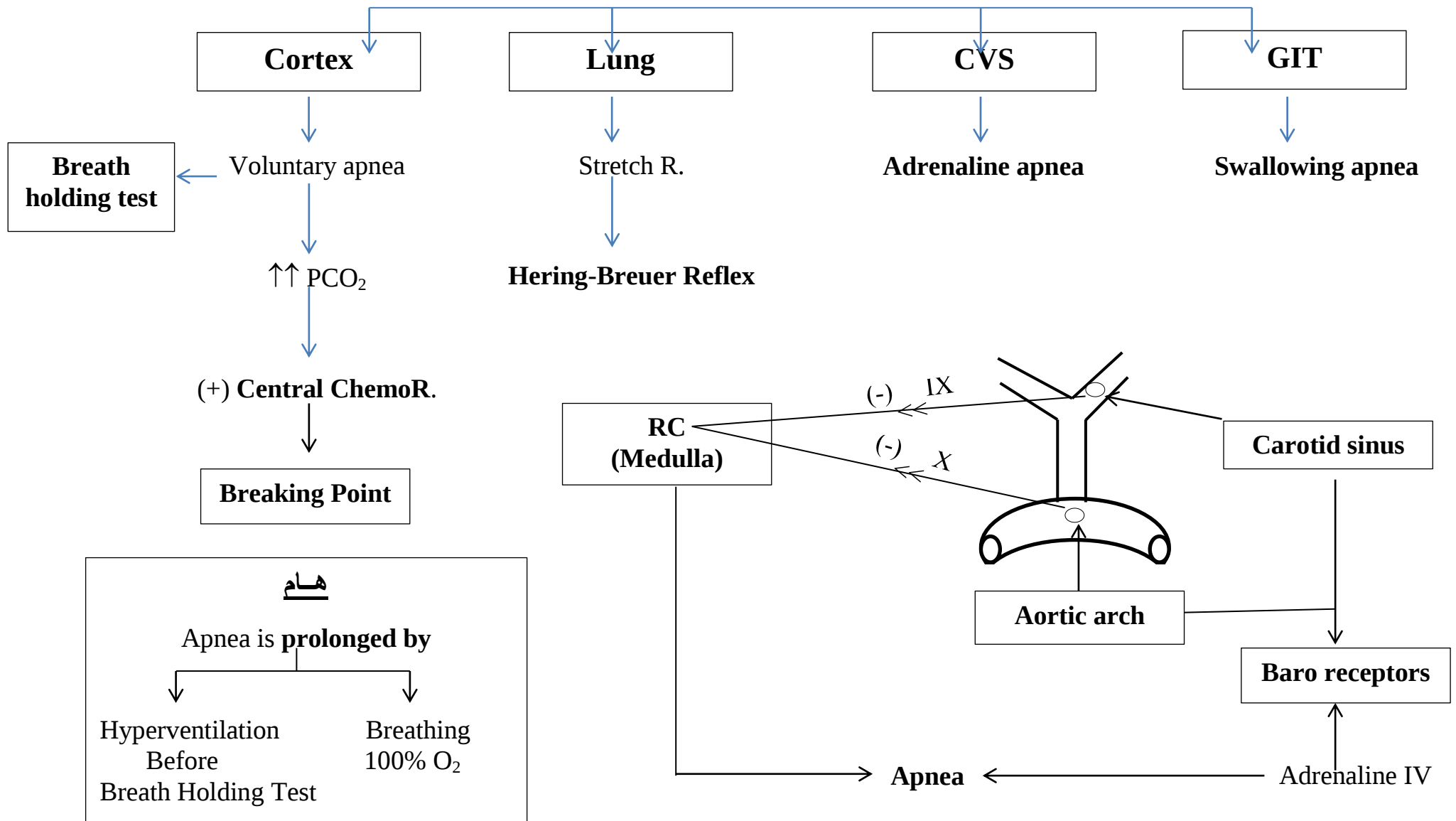
نفس Rapid shallow

Bradycardia

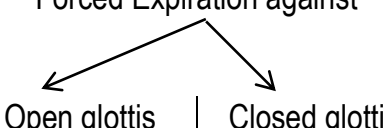
Hypotension

Pulmonary capillary

Non Chemical (Inhibitory) Regulation of Respiration د. محمد فايز



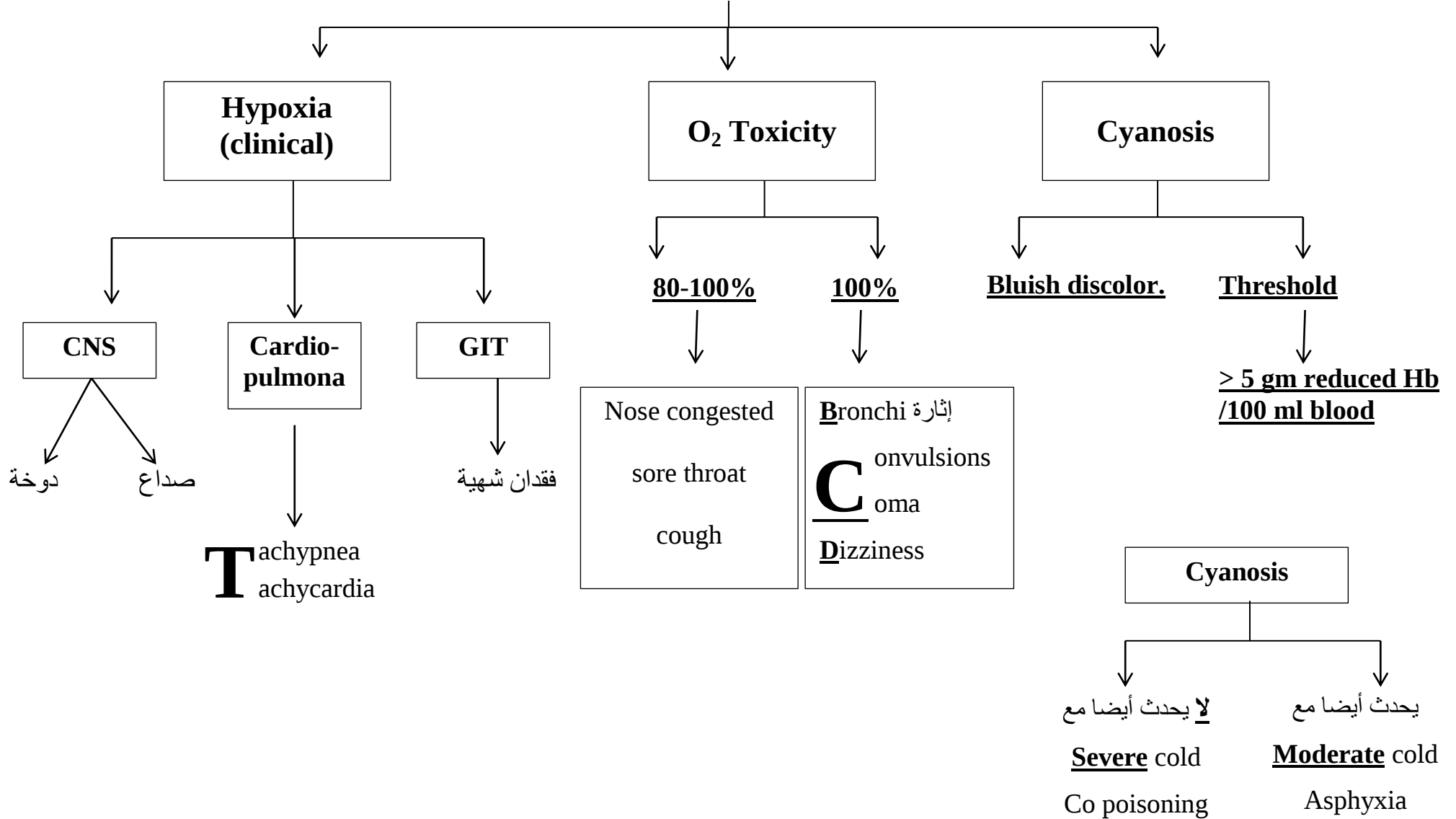
د. محمد فايز (Respiratory) Nonchemical Regulation of Respiration

	R e f l e x e s				Lung irritant receptors	J receptors	Chest wall receptors
	Sneezing	Coughing	Swallowing	Hering-Breuer			
Stimulus	Irritation of nasal mucosa	Irritation of respiratory mucosa	Stimulation of pharyngeal mucosa	Lung inflation	Irritant inhalation e.g. ammonia	Pulmonary congestion E demabolism	Stretch of chest wall muscles, tendons
Receptor	Irritant R (Nasal mucosa)	Irritant R. (Trachea, larynx, Bronchi)	Mechano R. (Pharynx)	Stretch R. (wall of bronchi/oles)	Irritant R. (wall of bronchi/oles)	Alveolar wall close to pulmonary cap.	Ms spindles of chest wall muscles
Afferent	Trigeminal	Vagus	Glossopharyngeal	Vagus	Vagus	Vagus	Intercostal
Response	Deep inspiration → Forced Expiration against 		Apnea	*inhibit insp. Center. *Start expiration	*Shallow rapid breathing. *Broncho const.	Shallow rapid breathing	*Inhibit Insp. Center. *Start expiration

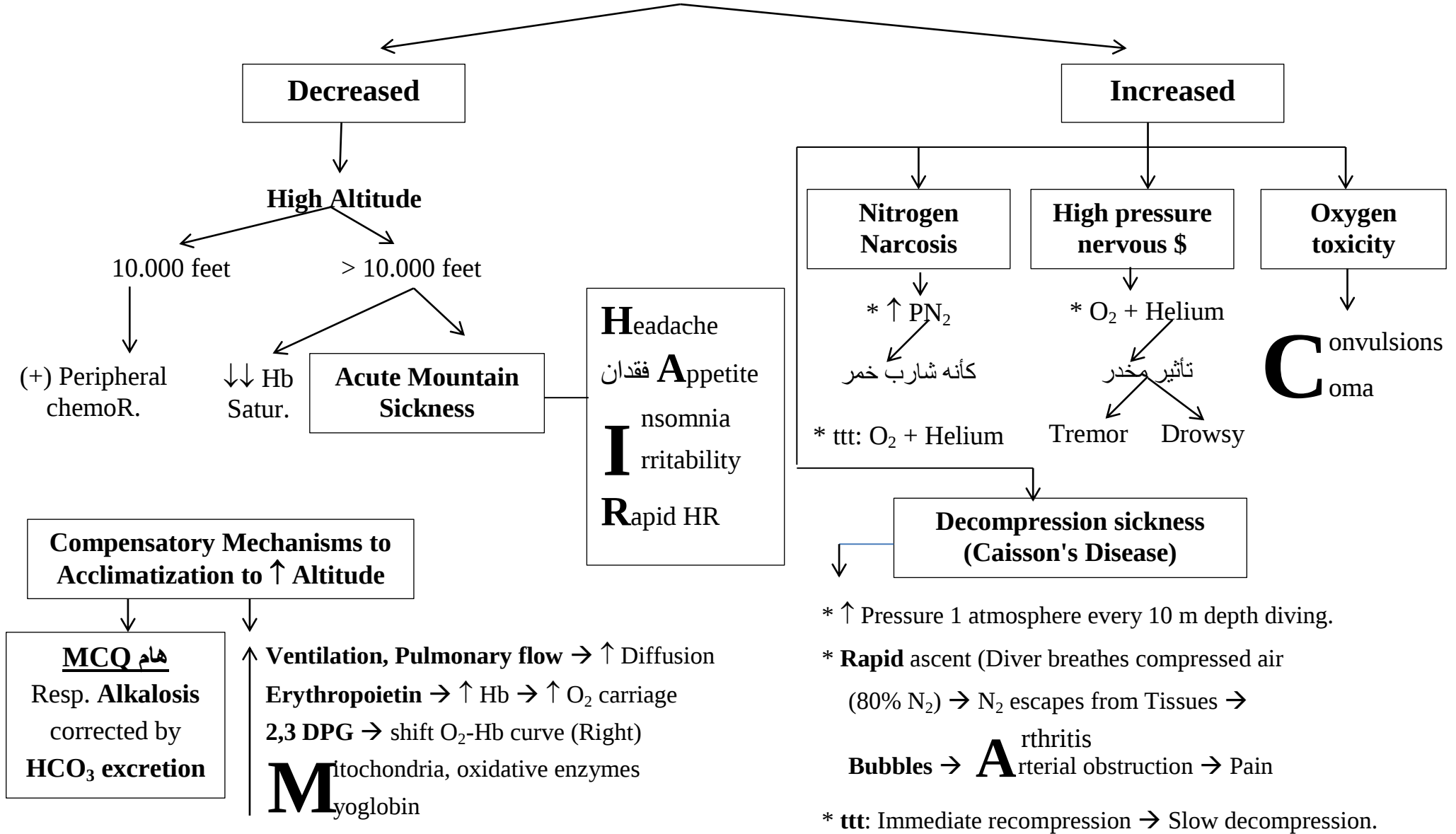
د. محمد فايز Hypoxia

	Hypoxic	Anemic	Stagnant	Histotoxic
Causes	1. $\downarrow PO_2 \rightarrow$ High altitude 2. Lung Diseases Hypoventil. $\downarrow\downarrow$ Gas Diffusion VA/Q Imbalance $\downarrow$ $\downarrow$ P neumonia ul. Congestion ul. fibrosis $\downarrow$ <u>Emphysema</u> P <u>Morphine</u> oliomyelitis neumothorax ulmonary (Obstructive) 3. Shunt of <u>Venous</u> blood into <u>Arterial</u> blood	1. Anemia 2. CO poisoning	General $\downarrow$ Heart failure Local $\downarrow$ Vascular spasm	Block of cellular Oxidative enzymes Cyanide Alcohol $\downarrow$ <u>Treated by</u> Nitrite, Methylene blue (react with cyanide) $\rightarrow$ Cyan Hb (Non Toxic)
PO₂	(V\$D) $\downarrow$	N o r m a l		
O₂ Therapy	Beneficial except "shunt"	L i m i t e d		Hyperbaric
Cyanosis	+	-	+	-

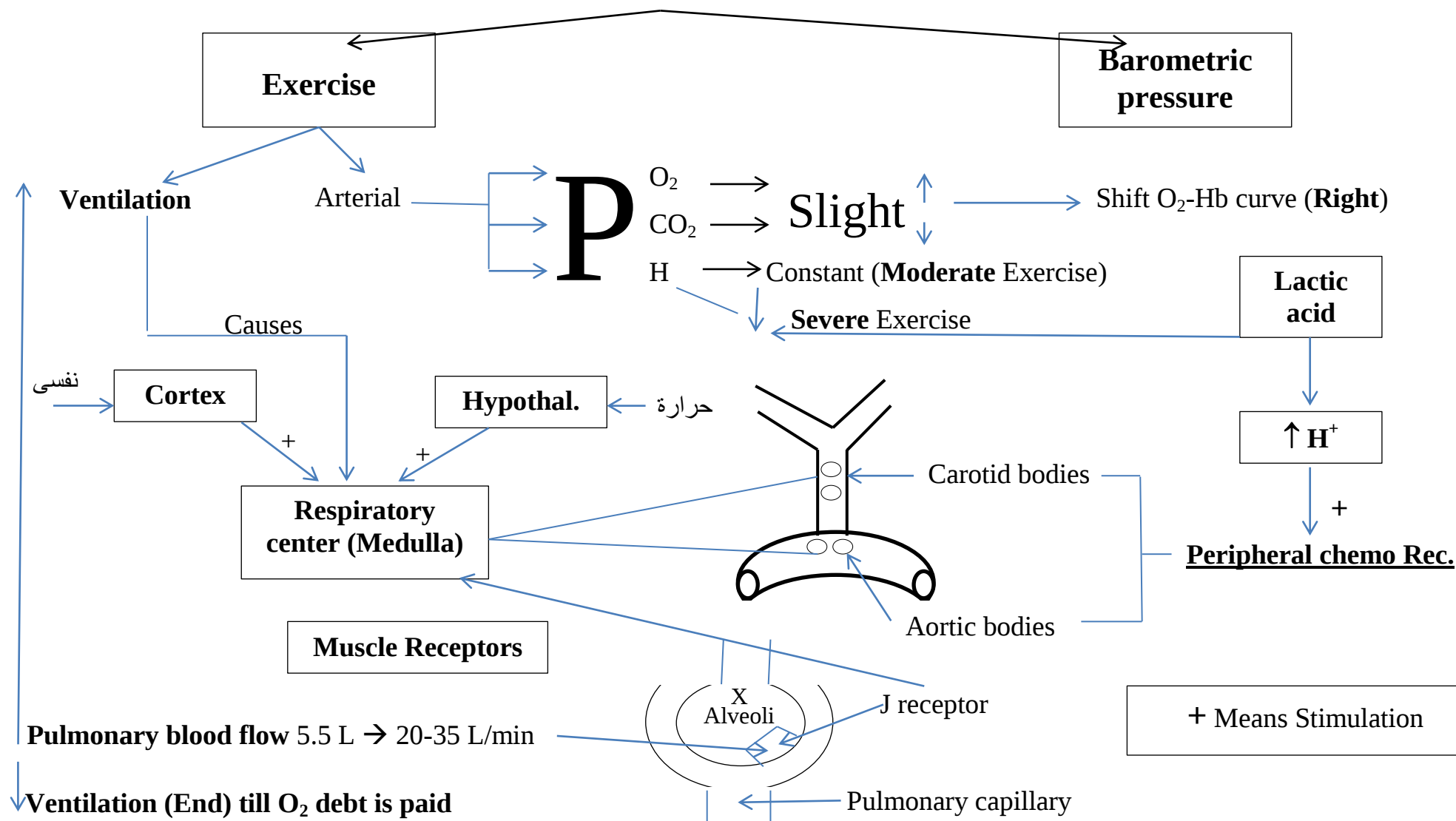
د. محمد فايز Notes



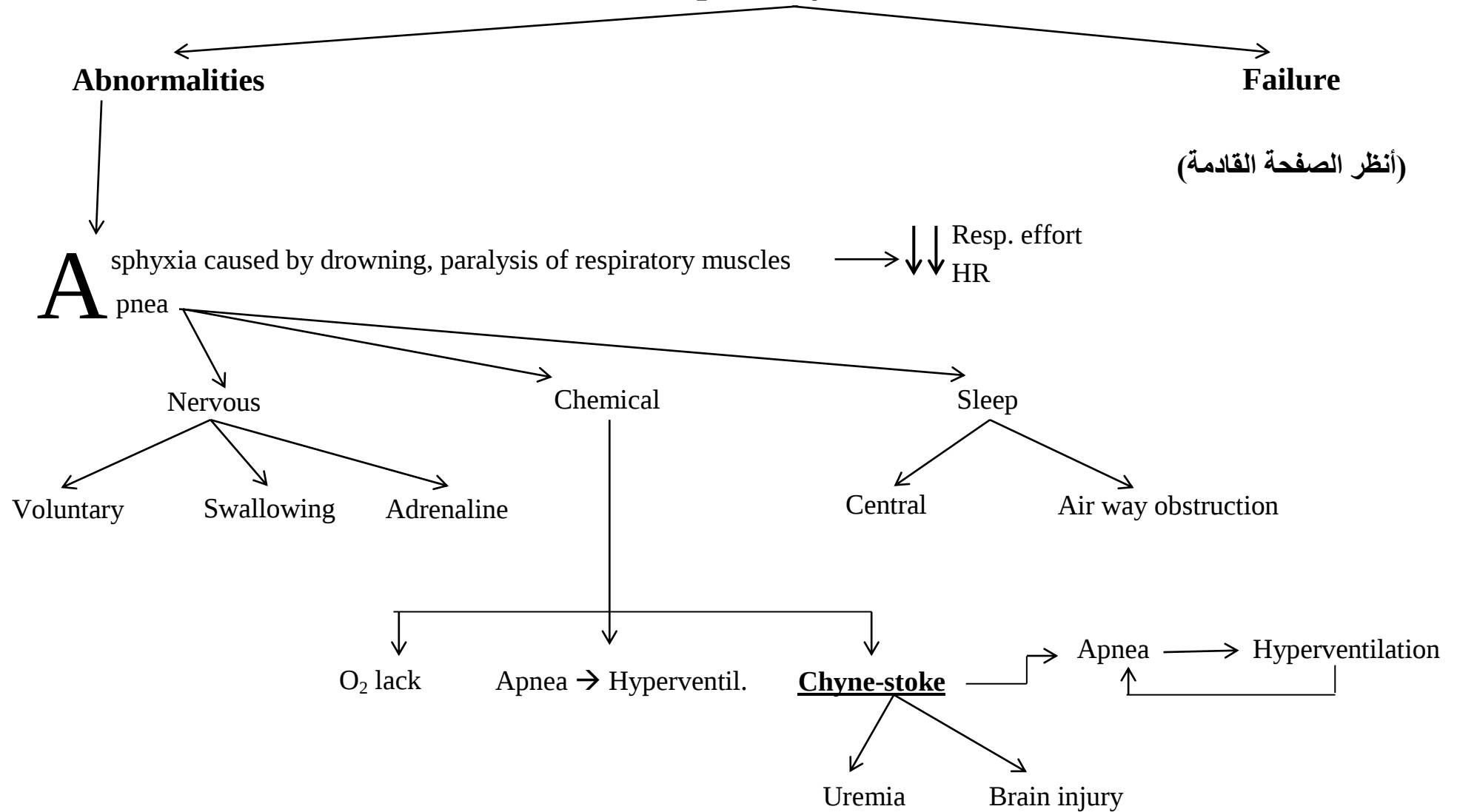
Barometric Pressure د. محمد فايز



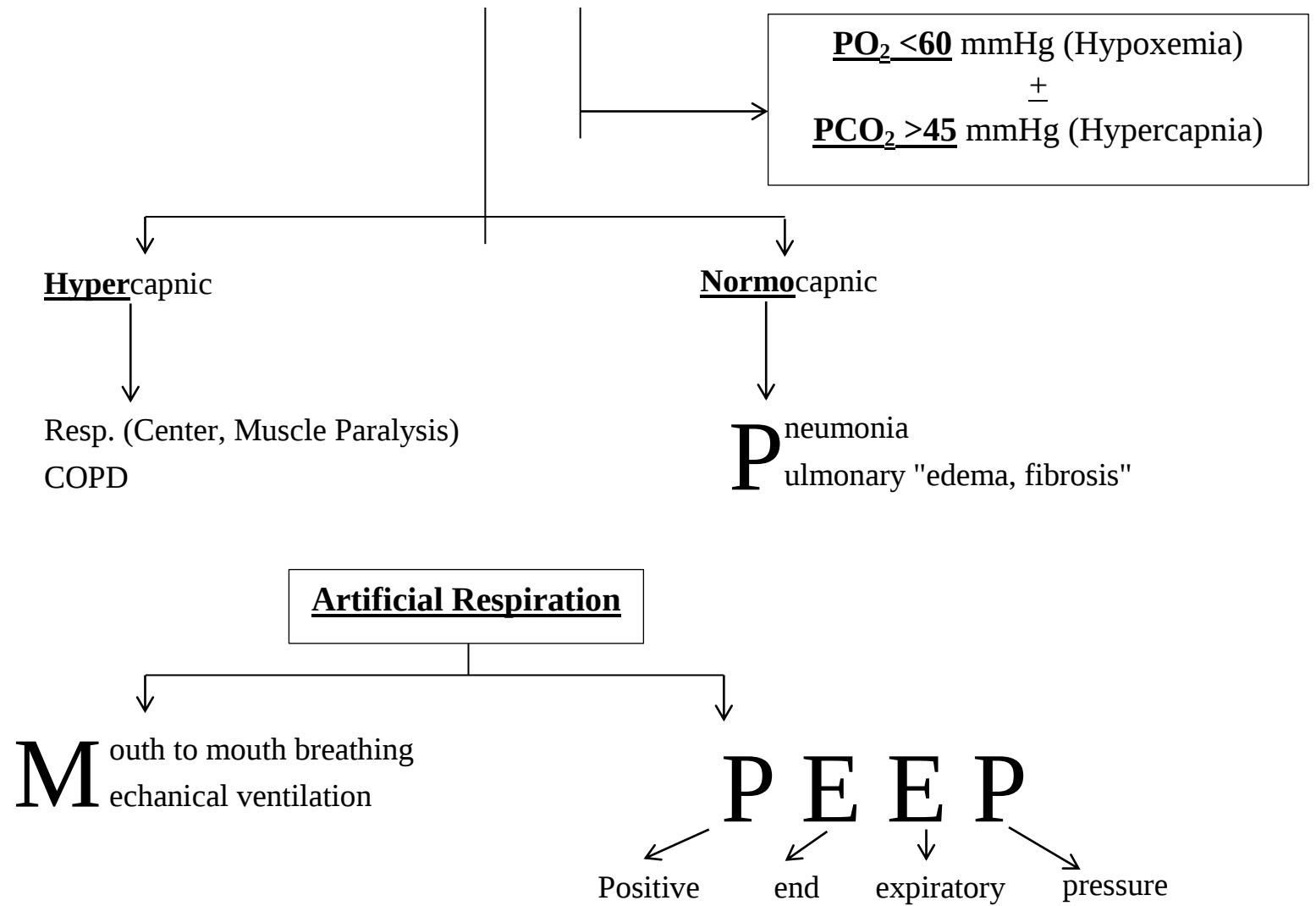
Respiratory Adjustment in Health, Disease د. محمد فايز



د. محمد فايز Respiratory



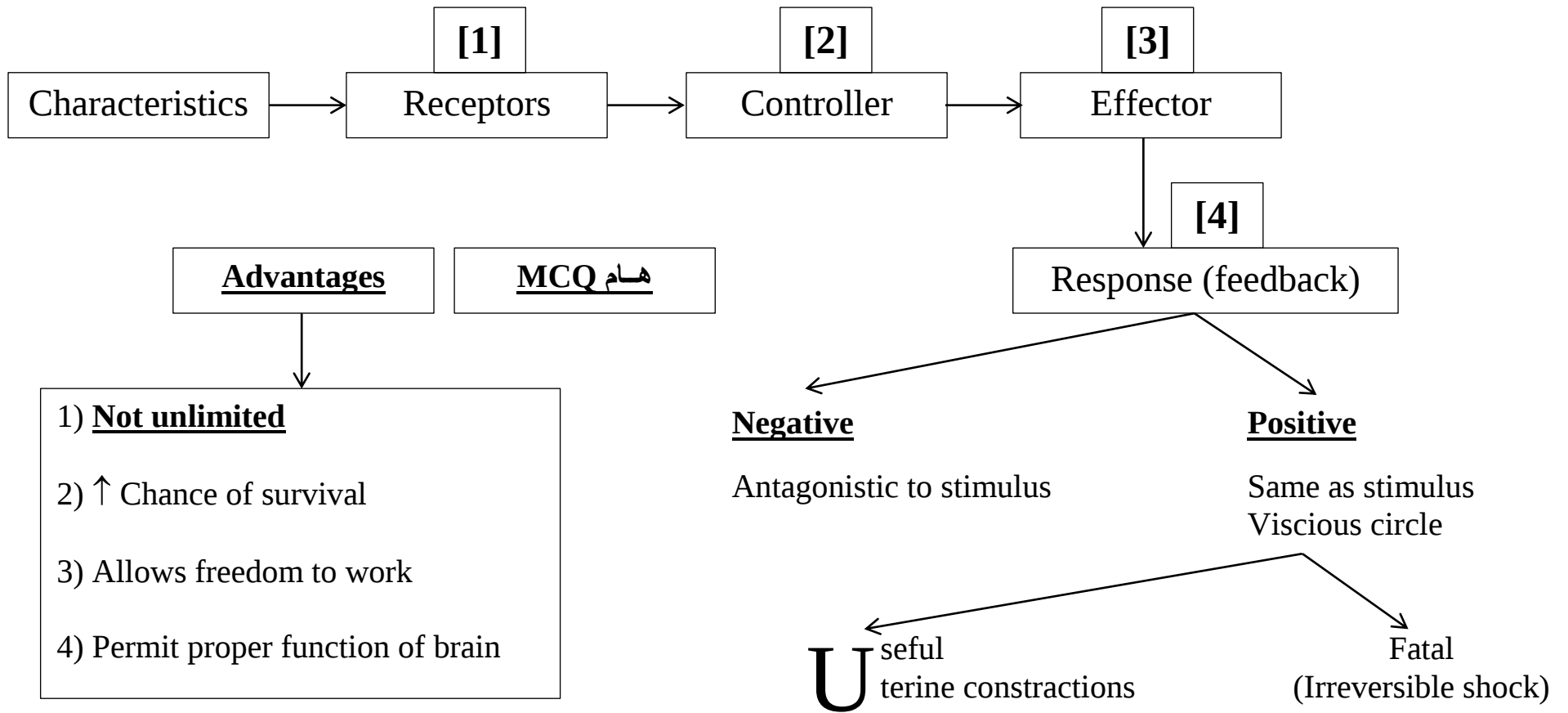
د. محمد فايز Respiratory Failure



Biophysics

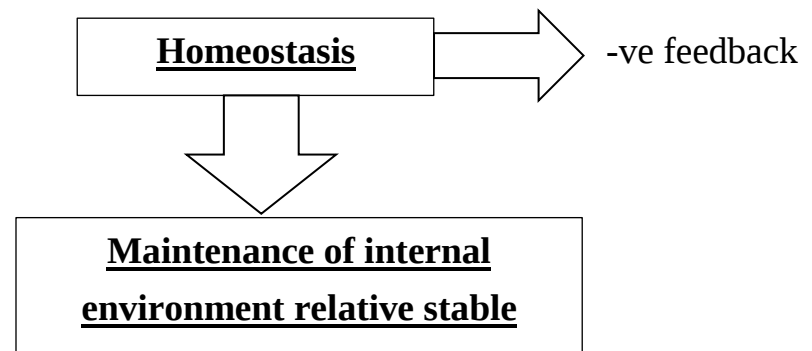
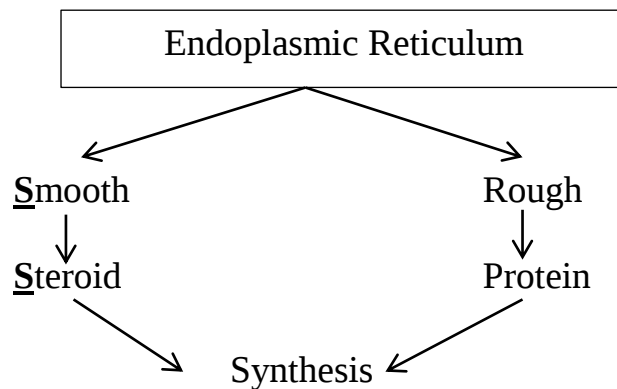
د. محمد فايز Homeostasis

Definition: Stability of internal environment



د. محمد فايز Pressure, Resistance

Carbohydrates	Lipid	Proteins
Receptors Blood group markers Identity markers Immune reactions	Barrier (Selective) Permeability Fluidity Membrane	Receptors Carriers ($\neq$ Pmps) Channels (Ion) Cell adhesion molecules Catalyze chemical reactions



Total Body Water د. محمد فايز

Decreases with age

Weight of adult male 70 Kg

Females: Less (↑ fat)

60%
===
42 L

28 L

ICF

14 L

ECF

ISF: 10.5 L

Plasma: 3.5 L

Volume of Injected Substance

===

$$\frac{\text{Amount injected} - \text{Amount removed}}{\text{Concentration}}$$

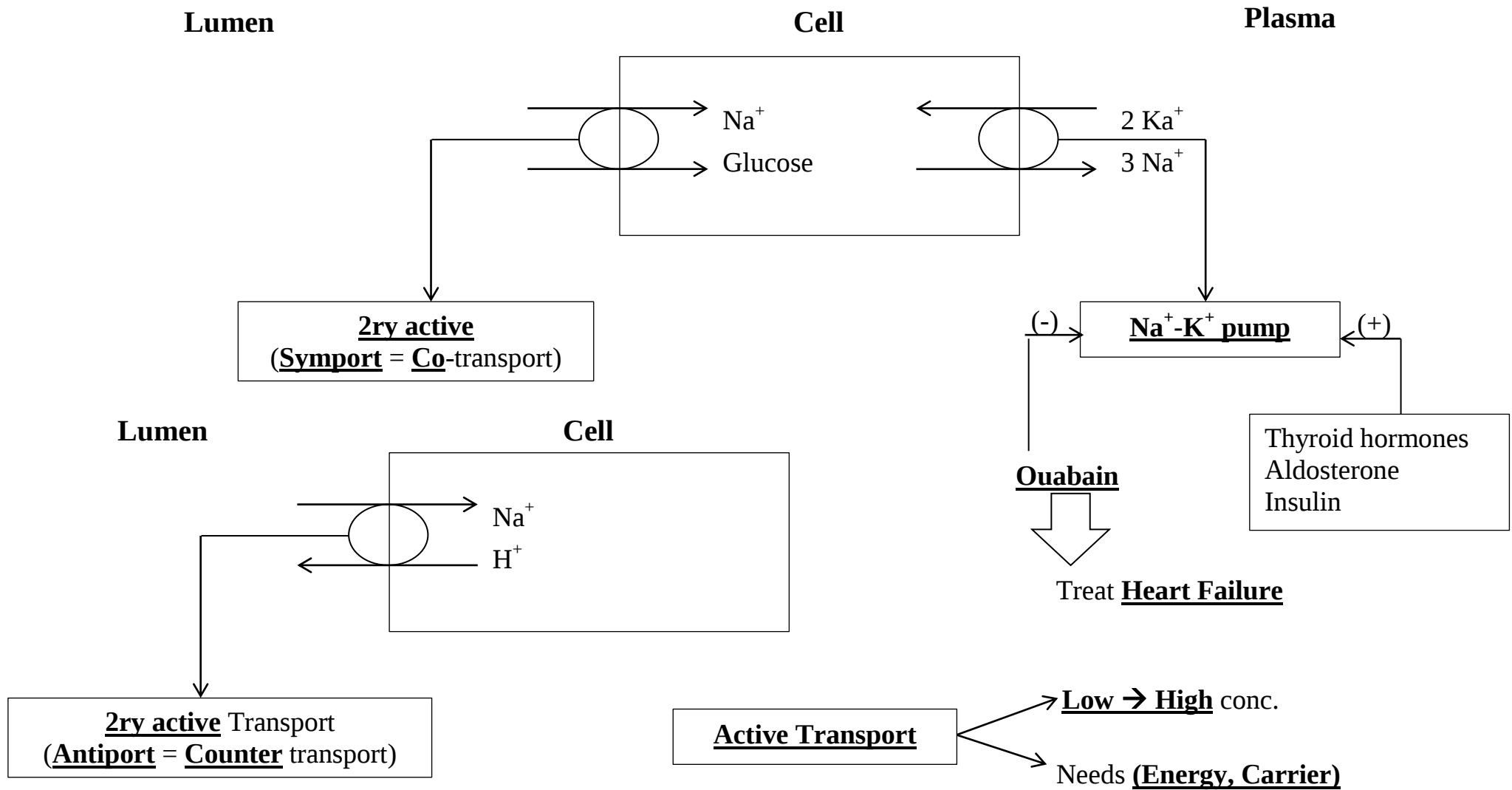
↑ ECF/ICF (Infant, Child)

More severe dehydration

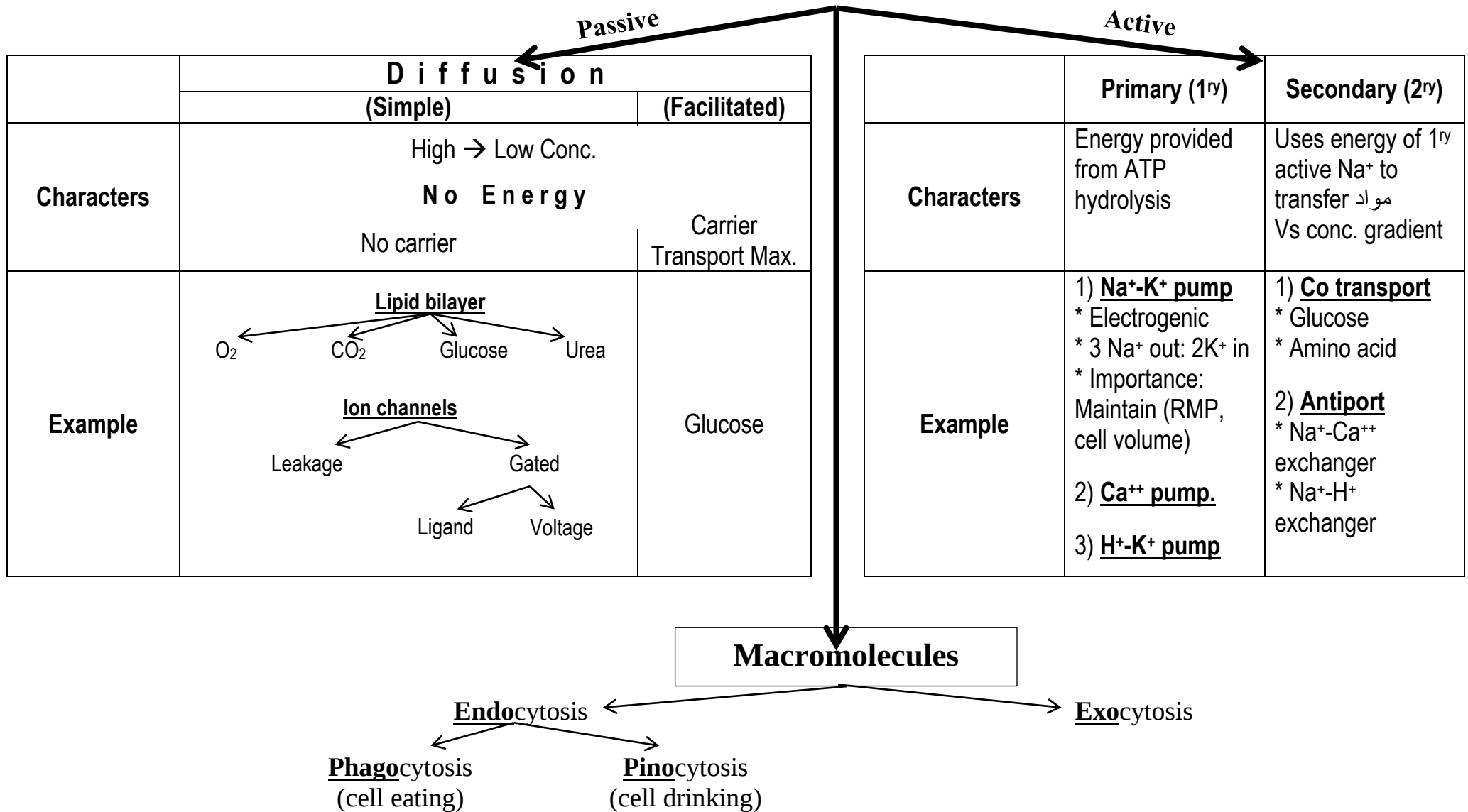
Proteins ICF > Plasma > ISF

	TBW	ECF	Plasma	ISF
Measured by:	* Labeled water a) Deutrium oxide b) Tritum oxide * Aminopyrine	I nulin isotopes T hiosulphate thiocyanate Sucrose, Mannitol	Evans Blue Albumin labeled with ¹³¹ I	Not measured Directly

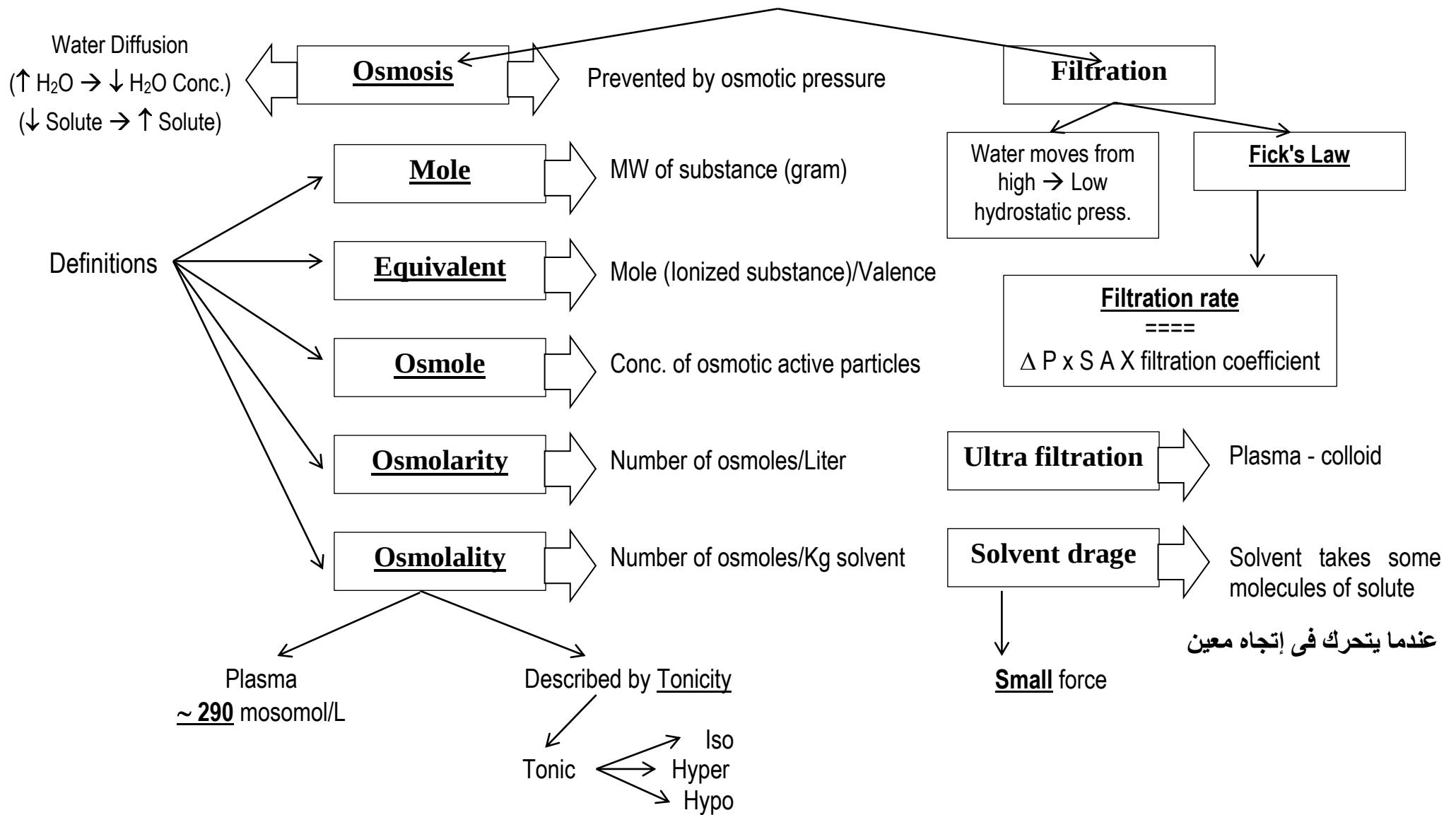
د. محمد فايز Transport



د. محمد فايز Transport

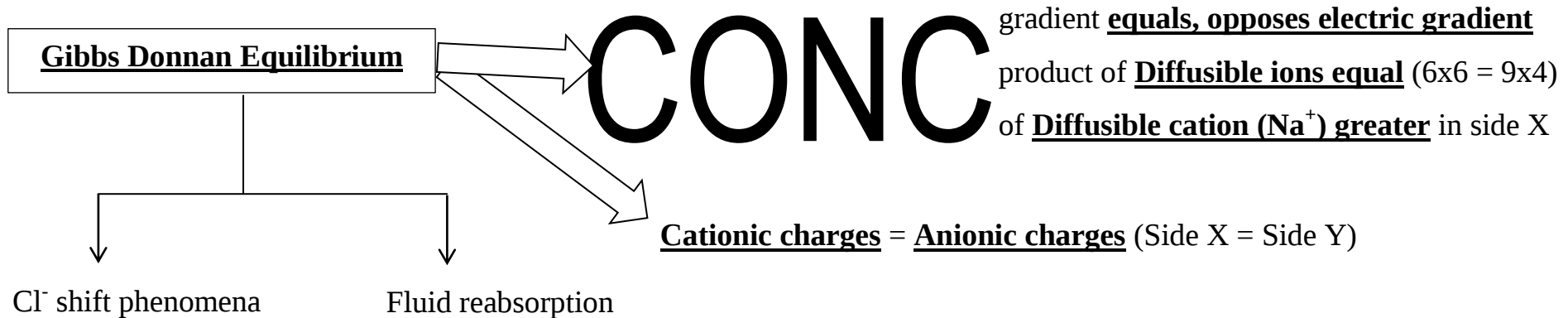


د. محمد فايز Movement of Water

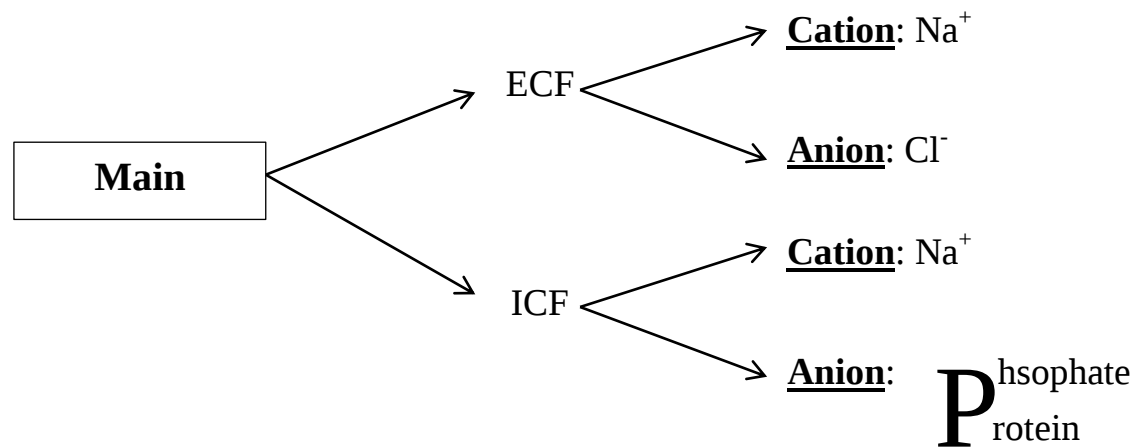
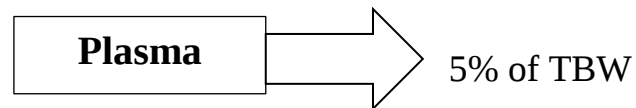
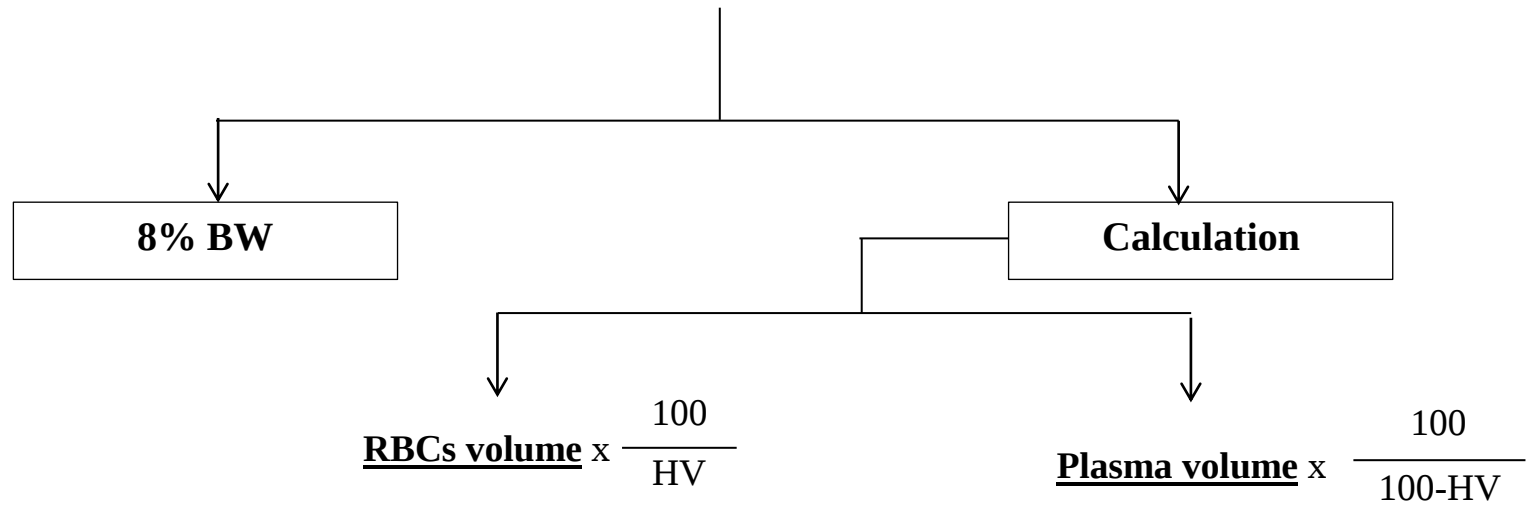


د. محمد فايز Non Diffusible Ions (Proteins)

Before equilibrium		At equilibrium	
Side X	Side Y	Side X	Side Y
5 Na ⁺	10 Na ⁺	9 Na ⁺	6 Na ⁺
5 Protein ⁻	10 Cl ⁻	4 Cl ⁻	6 Cl ⁻
		5 Protein ⁻	

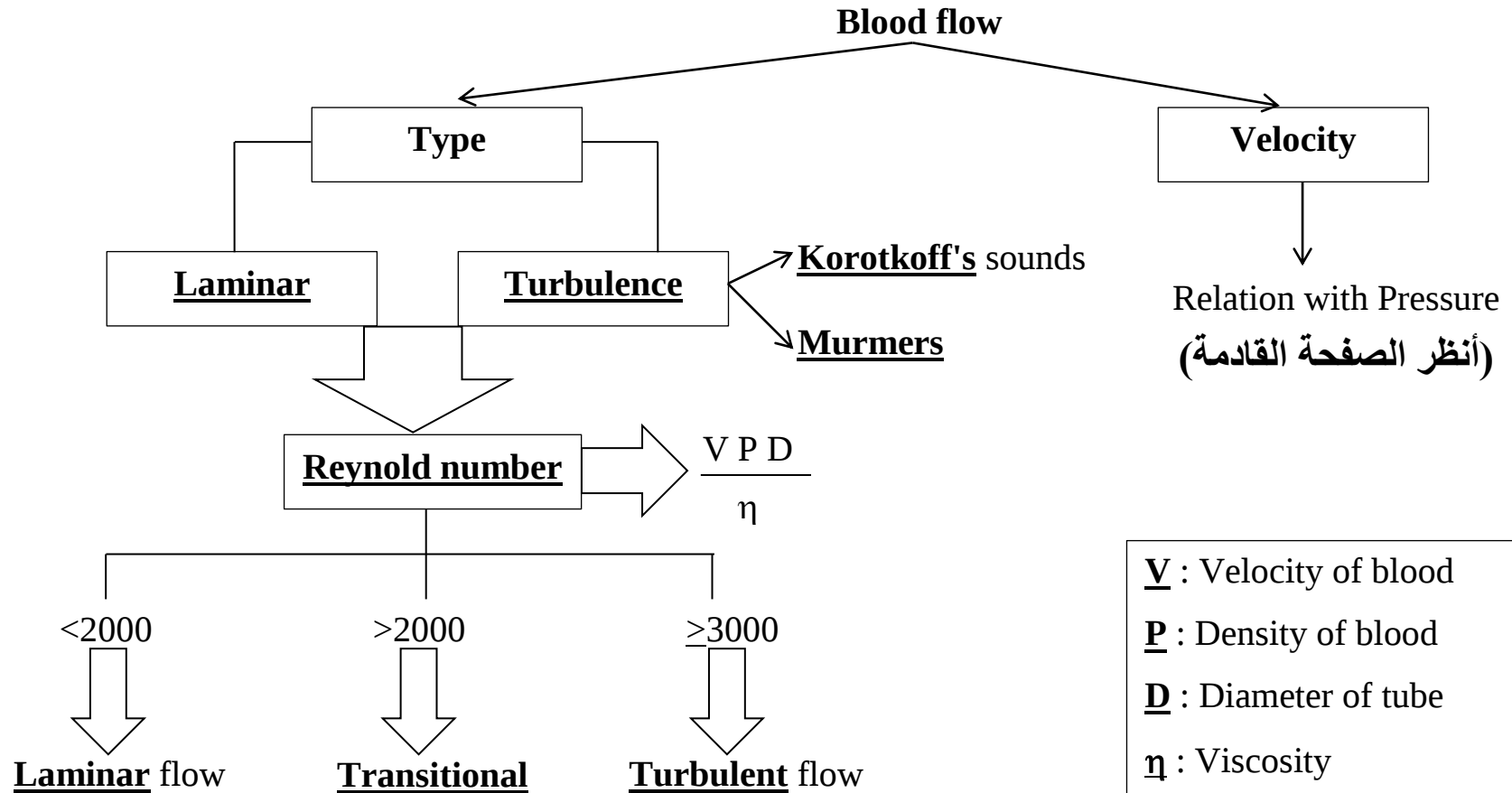


د. محمد فایز Blood Volume

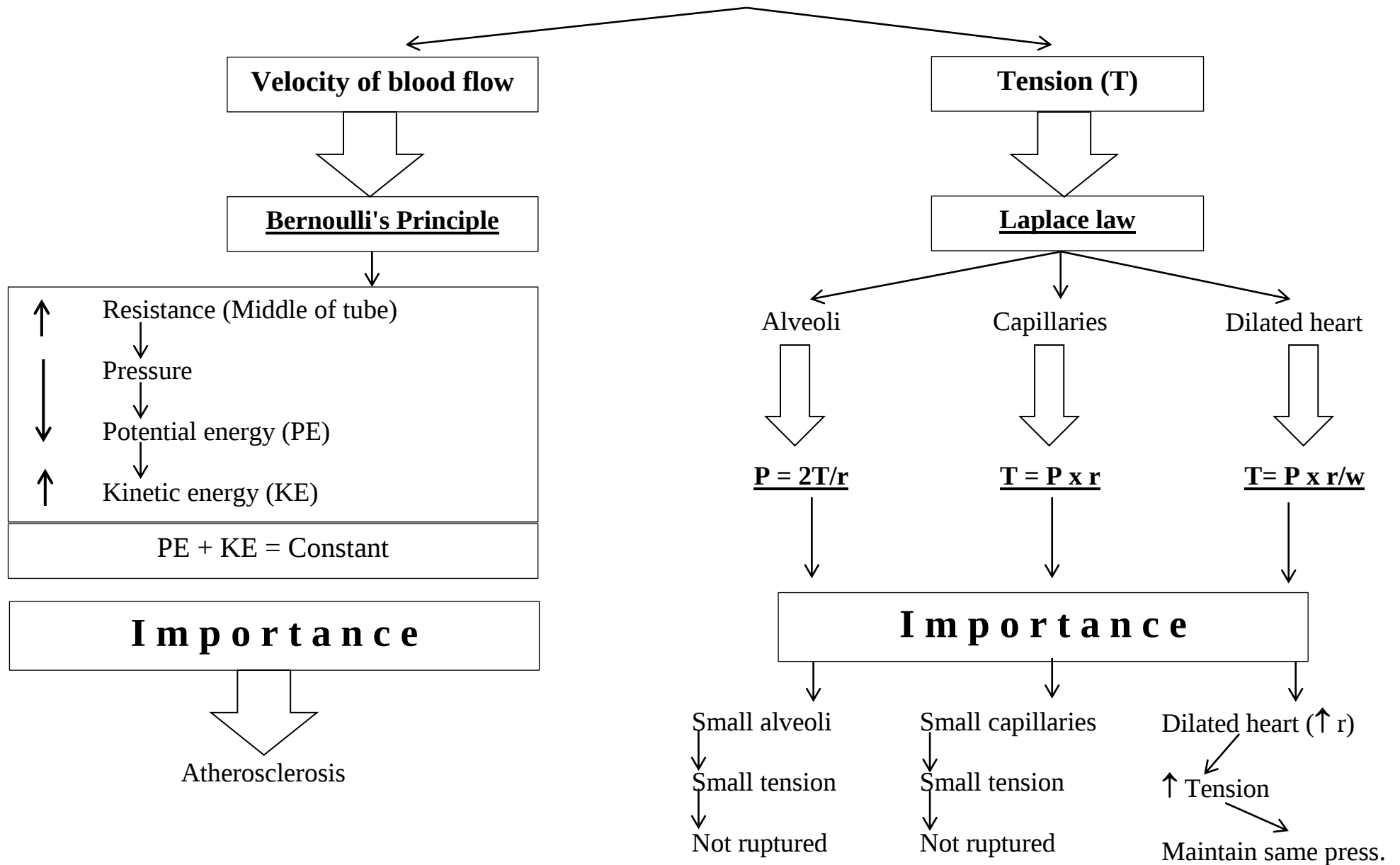


د. محمد فايز Physics Control Blood Flow

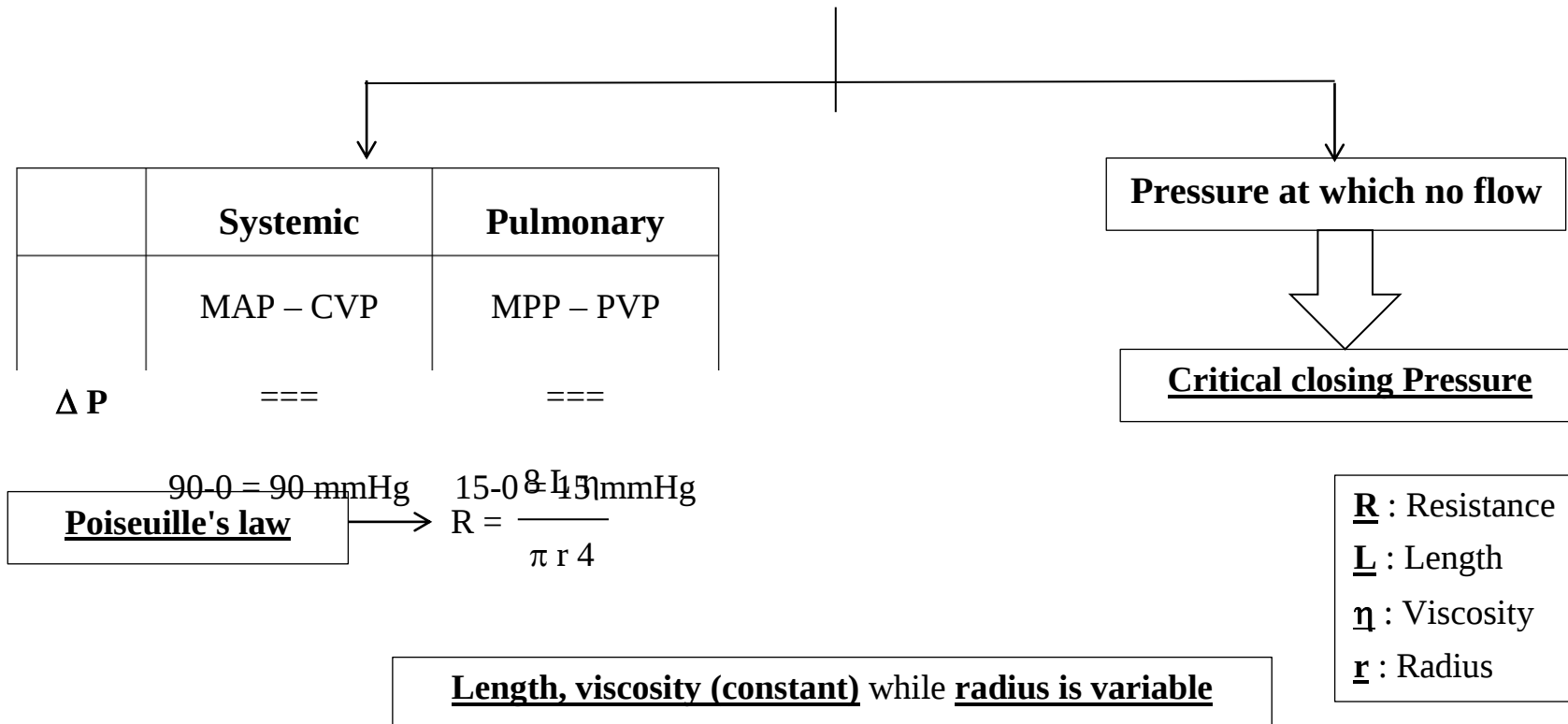
$$\text{Flow (F)} = \Delta P / \text{Resistance (R)}$$



Pressure relation with د. محمد فايز



د. محمد فايز Pressure, Resistance



MCQ

If radius is **doubled**, resistance will **decrease**:

- a) 2 Times.
- b) 4 Times.
- c) 8 Times.
- ✓ d) 16 Times.

Biostatistics

Data

وصف بدون أرقام

Qualitative

Quantitative

Nominal

Ordinal

Discrete

Continuous

لها اسم

لا يوجد ترتيب

e.g: Blood
groups

(A,B,AB,O)

ليس لها اسم

لها ترتيب

e.g: severe

head injury

يمكن تصنيفها حسب

الحالة

رقم بدون كسر

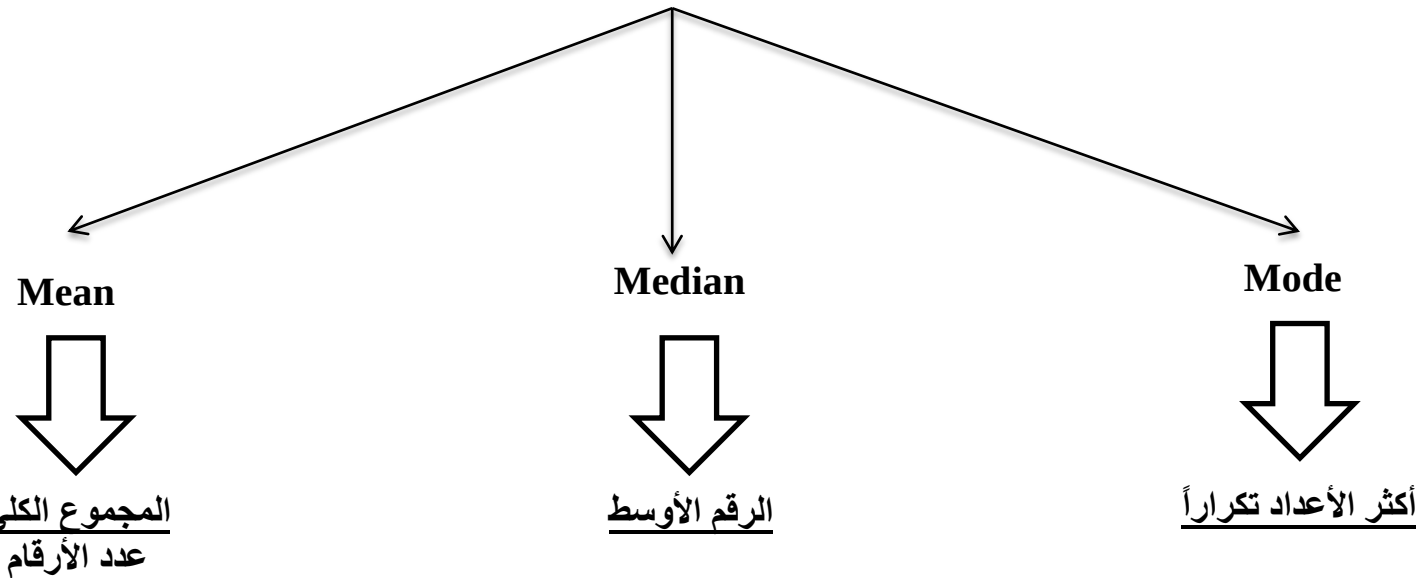
e.g: number of
Children (2-3)

رقم به كسر

e.g: weight
(58.5 kg)

Any Numerical Data (Age, Weight) is **Quantitative**

Central tendency (مجموعة أرقام يتم تمثيلها برقم واحد)



Advantage

- | | | |
|---------------------------|--|--|
| 1) <u>أفضل مقياس</u> | 1) <u>Not affected</u> by extreme values | 1) <u>Not affected</u> by extreme values |
| 2) <u>لا تحتاج لترتيب</u> | 2) Used for data | 2) <u>Nominal</u> data (<u>الأفضل</u>) |

Disadvantages values

- 1) Affected by **extreme**

ترتيب الأرقام
(Ascending or Descending)

N.B: 1) **Mode** is easily located by finding peak in Frequency Distribution graph

2) **Mode** may be > 1 e.g: 3, 4, 5, 6, 4, 6 (**4, 6**)

Dispersion Measures

Range

Mean Deviation

Standard Deviation (SD)

Variance

(Most Common)

يساوى أكبر رقم – أصغر رقم

Simple
ensitivity (**Not**)
cores (**Extreme Limit**)

الخطوات

- (١) إحسب Mean للأرقام
- (٢) إطرح Mean $(\bar{x})$ من كل رقم (x)
- (٣) إضرب ناتج طرح كل رقم فى نفسه
- (٤) إجمع الناتج من الخطوة الثالثة
- (٥) إحسب Mean للخطوة الرابعة

انظر مسألة الصفحة القادمة

$$\sqrt{\text{Mean Deviation}} = \text{SD}, \quad \sqrt{\text{SD}} = \text{Variance}$$

Central Tendency مسائل عن

Odd numbers

4, 5, 6, 4, 7

$$\text{Mean} = \frac{4 + 5 + 6 + 4 + 7}{5} = 5.2$$

$$\text{Median} = \frac{n + 1}{2} = \frac{5 + 1}{2} = 3$$

↓
Arrange numbers 1st (Ascending)

- 1) 4
- 2) 4
- 3) 5 ←
- 4) 6
- 5) 7

Mode = 4.

Even numbers

4, 7, 5, 12, 6, 10

$$\text{Mean} = \frac{4 + 7 + 5 + 12 + 6 + 10}{6} = 7.3$$

$$\text{Median} = \frac{3^{\text{rd}} + 4^{\text{th}} \text{ number}}{2} = 6.58$$

↓
Arrange numbers 1st (Ascending)

- 1) 4
- 2) 5
- 3) 6
- 4) 7 ←
- 5) 10
- 6) 12

Mode (لا يوجد)

Calculate Mean Deviation, Standard Deviation

x	$x - \bar{x}$	$(x - \bar{x})^2$
12	$12 - 9 = 3$	9
11	$11 - 9 = 2$	4
10	$10 - 9 = 1$	1
9	$9 - 9 = 0$	0
9	$9 - 9 = 0$	0
9	$9 - 9 = 0$	0
8	$8 - 9 = -1$	1
7	$7 - 9 = -2$	4
6	$6 - 9 = -3$	9

X : Number

$\bar{x}$: Mean = $81 / 9 = 9$

Mean Deviation = Sum of $(x - \bar{x})^2 / 9 = 28 / 9 = 3.11$

Standard Deviation (SD) = $\sqrt{3.11} = 1.76$

Calculate Mean Deviation, Standard Deviation

Table

Graph

Simple Frequency Distribution

Distribution of 20 child according to weight (kg)

14,12,22,15,16,20,18,19,23,11,16,22,24,23,26,14,26,17,26,27,24,18

	weight (kg)	Frequency	Relative Frequency %	Cumulative Frequency
1	10 – 14	3	15	15
2	15 – 19	7	35	50
3	20 – 24	6	30	80
4	25 – 30	4	20	100
	Total	20	100	

Complex Frequency Distribution

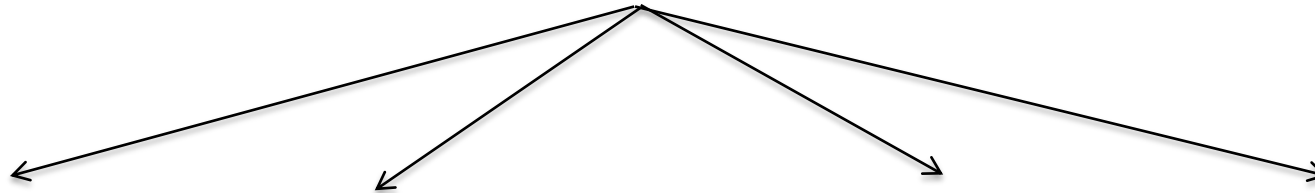
	Lung Cancer				Total	
	Case		Control			
	N	%	N	%	N	%
Smoker	15	75	8	20	23	38.33
Non Smoker	5	25	32	80	37	61.67
Total	20	100	40	100	60	100

Frequency: Range عدد الأرقام التي توجد في

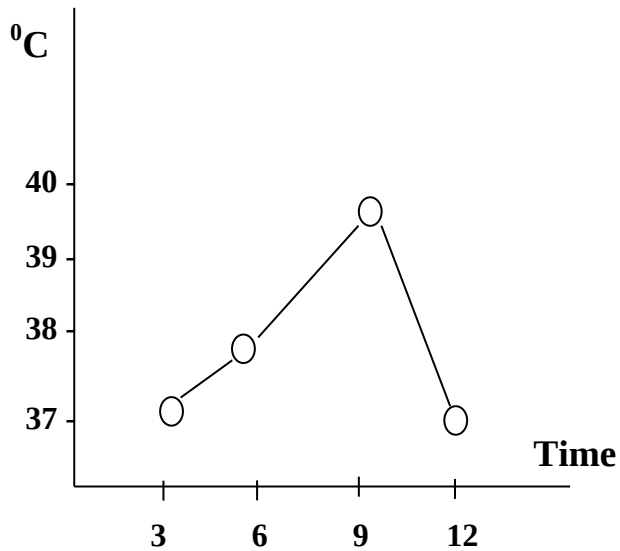
Relative Frequency %: $\frac{\text{Frequency}}{\text{Total}} \times 100$

Cumulative Frequency: جمع ما قبلها Relative Frequency

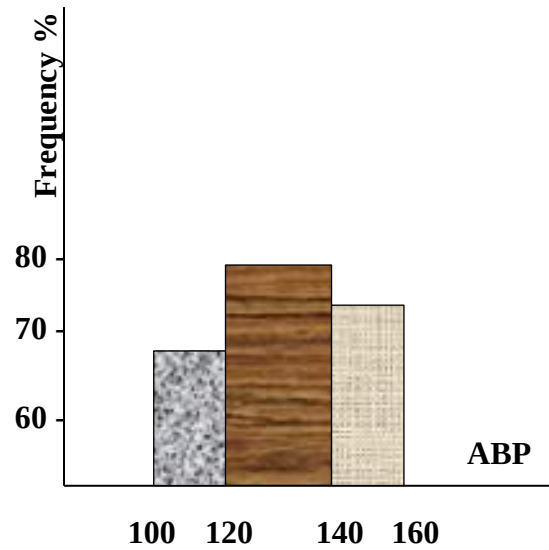
Graph



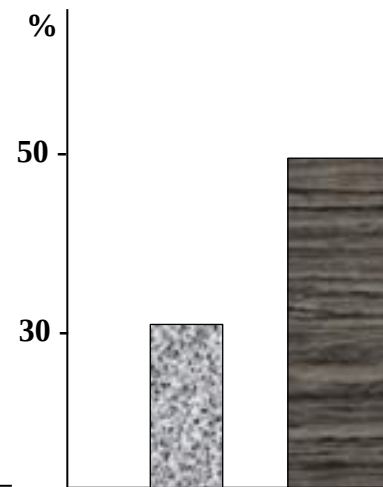
Line graph
(Temperature chart)



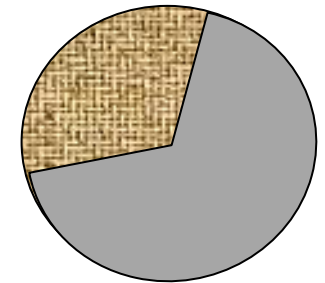
Histogram
(Continuous Variable)



Bar Chart



Pei Chart



Quantitative

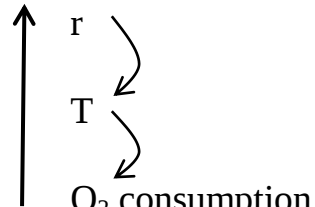
Qualitative

Collection

د. محمد فايز Compare

	Anemia	Polycythemia
Causes	1. Aplastic. 2. ↓ Iron, vitamin B12. 3. Hemolytic. 4. Hemorrhagic. 5. Anemia of renal failure.	<u>Primary</u> polycythemia (<u>Polycythemia vera</u>), e.g. Tumors of BM <u>Secondary</u> polycythemia (<u>Physiological</u>) e.g. High altitude
Hb	< 11 gm%	> 20 gm%
HV	30%	70-80% (1 ^{ry})
Blood indices	Detect its type	--
ESR	Increased except iron deficiency	Decreased
Cyanosis	Absent	Present
Pulse	Water hammer	--
Heart sounds	3 rd	--
ABP	--	↑ (↑ Viscosity → ↑ TPR)

د. محمد فايز Physiology Laws

	All or Non	Starling	Laplace
Definition	Threshold → firing level Maximal stimulus will give the same response	↑ Length of muscle → ↑ Force of contraction <u>Within limit</u>	Alveoli → $P = 2 T/r$ Capillary → $T = P \times r$ Dilated heart → $T = \frac{P \times r}{W}$
Significance	Action potential	<u>Skeletal, Cardiac Muscle:</u> * Tension $\propto$ force of contraction. * Best contraction at length 2.2 μ. * $> 2.2 \mu \rightarrow \uparrow$ force of contraction. <u>Cardiac Muscle</u> * $\uparrow$ Length = $\uparrow$ EDV = $\uparrow$ COP (RV) = COP (LV) = 5 L. * <u>Not applied</u> in <u>Normal</u> Heart. * <u>Applied</u> in (<u>Denervated</u>, <u>Transplant</u>, <u>Failing</u>) heart.	* <u>Alveoli, capillaries</u> Small radius (r) Small tension (r) Resist rupture * <u>Dilated heart</u> 

Physiology Laws

	Poiseuille	Fick's
	<u>Resistance (R)</u> = $\frac{8 L \eta}{\pi r^4}$	<u>Diffusion rate (DR)</u> = $\frac{\Delta P \times SA \times \text{Solubility} \times \text{Temp.}}{\sqrt{MW} \times \text{thickness}}$
Significance	Applied ↙ <u>Air way</u> ↘ <u>Blood vessels</u> <u>Radius</u> is <u>variable</u> factor which determines either <u>Resistance ↑ or ↓</u>	* Increased (<u>Exercise</u>) ↓ Δ P ↑ SA (open of closed alveoli) * <u>Decreased</u> e.g. <u>pulmonary edema</u> (↑ Thickness)

N.B.: **Mareys' law** (↑ BP → ↓ HR)

CVS Reflexes

	CNS Ischemic reflex	Cushing Reflex	Bezold-Jarisch Reflex
Stimulus	<u>MAP <50 mmHg</u> Decreased Blood flow to VMC <u>Normal CSF</u> <u>Brain Tumor</u>		* Myocardial infarction * Injection of: Veratridine Nicotine Serotonin
Response	Stimulate Ischemic Neurons (<u>Pressor area VMC</u>) Strong VC ↑ BP ↑ Blood flow to VMC Prevent death of brain neurons <u>(قبل ٣ دقائق)</u>		↓ HR ↓ BP

CVS Reflexes

	Baroreceptor reflex	Peripheral chemoreceptor reflex	Bain Bridge Reflex
Stimulus	Pulse pressure <u>fluctuations</u> ↓ Discharge (<u>sustained press.</u>)	<u>MAP <80 mmHg</u>	VR
Receptor	Aortic arch, carotid sinus	<u>Bodies</u> (Aortic, carotid)	<u>Type B "Atrial"</u> , Pulmonary
Afferent	IX, X	IX, X	X
Center	CIC, VMC	CIC, VMC	CIC
Efferent	Vagus	Sympathetic	X, Sympathetic
Response	* Stimulates CIC → ↓ HR * Inhibits VMC <pre> graph TD A["* Inhibits VMC"] --> B["Vaso-dilatation"] A --> C["veno-dilatation"] B --> D["↓ BP"] C --> E["VR EDV SV COP"] E --> D </pre>	* Stimulates CIC → ↑ HR * Inhibits VMC <pre> graph TD A["* Inhibits VMC"] --> B["Vaso-constriction"] A --> C["veno-constriction"] B --> D["↑ BP"] C --> E["VR EDV SV COP"] E --> D </pre>	<pre> graph TD A["X"] --> B["↓ CIC"] B --> C["Tachycardia"] D["Sympathetic"] --> E["↑ SA node"] E --> C </pre>

Compare CO₂, O₂

	CO ₂	O ₂
No Exchange	Nose → Terminal Bronchioles (Dead Space)	
Transport	90% (HCO ₃) 5% (Carbamino compound) 5% (Hb)	Hb, Myoglobin
% Hb Saturation	2.5%	97.5%
Dissociation curve	Linear	Sigmoid
↑ release	<u>Shift to right</u> From tissues → Hb unloads O ₂ (Bohr effect)	From Hb → ↑ Hb affinity to CO ₂ (Haldane effect)
Effect on ventilation	Receptors stimulated: (Central: Main), Peripheral: Weak Increased > 5% → ↑ Ventil. 3-4 Times > 70-80% → RC depression Decreased → Apnea 	Receptors stimulated: Peripheral chemo R. only ↓ PO ₂ < 60 mmHg → ↑↑ Ventilation
Respiratory failure	Hypercapnic	Hypoxemia (Normocapnic)
Effect on BV	VD	Lack causes VC
Effect on cerebral circulation	VD	Physiological: No or limited effect Pathological: PO ₂ (30 mmhg) → 2 CeBF.

Compare CO₂, O₂

	CO ₂	O ₂
<u>Content</u>		
Arterial	48 ml/100 ml	19.8 ml/100 ml
Venous	52 ml/100 ml	--
<u>Pressure</u>		
Arterial	40 mmHg	100 mmHg
Venous	46 mmHg	40 mmHg
MW	High	Low
Solubility	24 Times more	
<u>Diffusion coefficient</u>	20 Times more	
<u>VA/Q</u>		
Local Homeostatic	↓ CO ₂ → Bronchoconstriction	↓ O ₂ → VC
Imbalance	↓ CO ₂ → Shift ventilation away from poor perfused alveoli	
<u>Exchange</u>	Alveoli	

Note the difference

Boyle's effect	Boyle's equation
↑ CO₂ release ↓ Hb affinity to O₂ (Hb unloads O₂)	Measures dead space $\frac{\% \text{ CO}_2 \text{ expired} - \% \text{ CO}_2 \text{ Alveolar air}}{\% \text{ CO}_2 \text{ expired}} \times 500$ $\frac{6\% - 4\%}{6\%} \times 500 = \simeq 150 \text{ ml}$
Active hyperemia	Reactive hyperemia
↑ Blood flow to tissue	Blood flow returns to tissue after <u>temporary occlusion of BV</u> e.g. 1) Skeletal muscle. 2) Cardiac muscle

Ficks law	Ficks principle
<u>Diffusion rate (DR)</u> === $\frac{\Delta P \times SA \times \text{Sol.} \times \text{Temp}}{\sqrt{Mw \times \text{Thickness}}}$	<u>Measures COP</u> === $\frac{\text{Tissue O}_2 \text{ consumption}}{\text{A-V difference}}$ = 5 L/minute
Local potential	Local response
• Subthreshold • Graded, summated • NO A_{RP} LL or none law • Decremental conduction • Site: Receptor	When cathode (+) produce 7 mv ↓ Some voltage gated Na⁺ channels open ↓ Magnitude of depolarization greater than expected.

Orthodromic: Same direction

Antidromic: Opposite direction